THE
VETERINARY JOURNAL

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Editorial

POULTRY HUSBANDRY.

THE present position with regard to the poultry industry is that of all the branches of agriculture carried out in this country the value of this industry comes easily first. It is estimated that nine years ago the value was only one-fifth of that of the present day. The rapid increase in the poultry world has necessitated much research and study on the problems vital to its growth; this has resulted in many workers being engaged in all types of researches, the results of which have been applied to the industry, and have contributed largely to its successful growth. Evidence that much information in connection with poultry husbandry is accumulating all the time can be obtained by a perusal of the reports and papers presented at the various poultry conferences which are held from time to time. Recently the Annual Harper Adams College Poultry Conference was held, and still more recently the Fifth World's Poultry Congress met in Rome; at both meetings papers of the utmost importance to the poultry industry were presented and discussed. Poultry husbandry has also been the subject of papers and discussions during the past year at the Annual Congress of the Royal Sanitary Institute and at the Annual Congress of the N.V.M.A. In this number of THE VETERINARY JOURNAL, in addition to original articles, there is reproduced a selection of papers presented at these Congresses, as well as several abstracts of other papers, the reading of which will give an indication of the important types of work relating to poultry which are being carried out at the present time in this and other countries.

In connection with the study of these subjects, it is very gratifying to note that members of the veterinary profession are commencing to take an active part, but

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we feel that the profession as a whole does not realise fully the important position occupied by the poultry industry to-day. It is true that veterinary research workers have shown how to deal with several of the infectious diseases prevalent among our flocks, but there is still a long way to go before we can claim that we have poultry diseases under real control. The veterinary profession, by its training in animal diseases generally, should be in the position to advise on, and control, poultry diseases ; but there appears to be a general lack of interest by the profession as a whole, with the result that the work is apt to be undertaken by others who cannot be in the position to do the work as competently as we ought to be. The industry has grown so quickly that we have not yet grasped its magnitude and importance, and unfortunately we are not yet in the position to undertake the control of the diseases of birds. It may be that the value of the small flocks in which diseases appear do not merit the calling in of the veterinary practitioner, but the work should be undertaken by those of us who have specialised in the study of diseases of birds. There is an opening of considerable value for veterinary surgeons in this field, and we feel that more training in poultry husbandry generally, and poultry disease in particular, should be found in the various veterinary teaching institutions ; so that those who desire to specialise in this branch of animal husbandry can have a good foundation on which to build their later specialised knowledge. The latter can only be acquired by first-hand information obtained from practical experience. The actual investigation of diseases in so far as their cause, prevention, etc., are concerned, must always be in the hands of the research workers, but the controlling of disease, no matter from what cause, and the application of the results of the studies of research workers should be in the hands of veterinarians who have specialised in the subject. Until the veterinary profession takes the matter up seriously and realises that it is the duty of its members specially trained in poultry husbandry by teaching and practical experience, to deal with poultry subjects in the field, that bugbear—disease—which limits the extent to which any animal industry can develop, will not be met to the greatest extent, but will still continue to be dealt with in a manner entirely out of proportion to this valuable industry.

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General Articles

ABSTRACTS OF SOME PAPERS READ AT THE FIFTH WORLD'S POULTRY CONGRESS, ROME, 1933.

(A) FOWL PARALYSIS.

(I) **Research on Fowl Paralysis and Allied Conditions.** H. P. BAYON, Cambridge University, England.

FOWL paralysis is widespread in Great Britain, but its relative incidence is not frequent; where heavy mortality is reported it is found that complicating diseases are present. It has been shown, by examining flocks in successive years, that the disease can be parentally transmitted through the egg, and that it occurs particularly in the progeny of certain stocks. The diagnosis of the condition is sometimes only possible after critical and accurate microscopy of the peripheral nerves and internal organs, including bone marrow. External symptoms may be misleading, as they occur in several different morbid conditions. Microscopical examination of lesions will differentiate between the different forms of paralysis, *e.g.*, caused by parasitic infestation, nutritional errors, etc. Artificial reproduction of the full clinical disease is not easy, though the transmission of simple lymphoma by inoculation has been accomplished in a few instances. The author holds that neuro-lymphomatosis results from the effect of two pathogenic elements, neural lesions originally induced by nutritional errors in the early life of the bird, and then an additional proliferation of cells of the lymphoidal series which settle in these damaged nerves. The prevention of the disease should be based on the correct diagnosis in the tainted stock, which should be gradually culled out. Spontaneous cures occur, especially where green food is abundant. Such "cured" birds may remain permanently lame, or may exhibit a permanently dropped wing, and their nerves when examined show chronic or quiescent lesions. The author was able to show that the forcible feeding of affected birds with fresh lettuce leaves was successful in curing twenty-eight out of seventy-two birds treated. In the cured birds the presence of typical lesions was confirmed by microscopic examination of the nerves.

(II) **Is there any possibility of limiting Fowl Paralysis?** G. LERCHE, Breslau, Germany.

Fowl paralysis is specific to the fowl, and is accompanied by inflammation of the central nervous system and of the peripheral nerves, producing phenomena of paralysis. It is caused by a specific agent of variable virulence, and as a rule causes infection only in chicks up to the age of fourteen weeks. The incubation period varies from two to seven months. Natural infection is caused by contact with diseased fowls and by carriers of the specific infecting agent. Cured hens

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may transmit the infecting agent to the egg, and the disease is generally spread by the sale of young stock. The disease is found in nearly all countries, and on infected farms may cause losses up to 30 per cent. In diagnosing the disease conditions caused by vitamin deficiency and parasitic infestations are important. Diagnosis is facilitated by a consideration of clinical symptoms, the course of the disease, post-mortem findings, and particularly histological examinations. The author makes the following suggestions for its control :—*Healthy Stock*.—Care in the purchase of young stock and care in rearing stock from other breeders. *Infected Stock*.—Careful culling of all newly purchased stock. Killing all paralytic birds. Raising chicks entirely separated from the rest of the poultry on the farm. The introduction of pullets only after they have begun to lay.

(III) **Infectious Paralysis of Fowls observed in Lombardy.** G. VIANELLO, Milan, Italy.

The author describes an outbreak of fowl paralysis on a large poultry farm where about 3 per cent. of the birds between five and nine months' old were affected. The symptoms were those of more or less acute paresis and paralysis of the extremities, localised chiefly in the lower part of the legs. Lesions consisted of a thickening and greyish coloration of the ganglia and the sacro-lumbar and axillary nerve plexuses, as well as the associated nerve bundles. Histologically, in all parts of the nervous system was a characteristic cellular infiltration (lymphocytes, plasma cells, etc.) in direct correlation with the appearance of the macroscopic lesions. Ætiologically there was no connection between the disease and vitamin deficiencies, nutritional disturbances, or parasitic infestation. The author was unable to transmit the condition experimentally. He thinks the disease was probably introduced by foreign cockerels bought for breeding purposes. A method of preventive vaccination is discussed.

(IV) **Fowl Paralysis (Neuro-lymphomatosis) in England.** T. DALLING and G. H. WARRACK, Beckenham, England.

This is a description of the disease as it occurs in England. The authors refer to the work of Galloway, who described the disease in 1929, and to Bayon's demonstration of the identity of the disease in England with range paralysis in America. Descriptions of the characteristic symptoms, lesions, and histological findings are given, and it is pointed out that the typical types of cells associated with the disease may be found in tissues other than those of the central or peripheral nervous systems, causing obvious macroscopic lesions of varying size. The occurrence of the disease on a farm can usually be traced to the importation of hatching eggs, young stock, or breeding stock from an infected farm.

The true cause of the disease is not known, though certain features of the condition are in keeping with a virus infection. The authors were able to set up the disease by the injection of young birds with emulsions of nerves, spleen and lungs taken from affected birds Their most recent experiments resulted in the development of the typical disease in 19 of the 70 birds injected, *i.e.*, 27 per cent.,

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but at the same time five, or 3.2 per cent., of the 156 untreated control birds of the same stock became affected. Further, it was found that the total death-rate during the period of observation among the inoculated birds was 38.5 per cent., while in the uninoculated group 8.5 per cent. died during the same period.

The authors express the opinion that "the likely explanation of our transmission results is that the whole stock was susceptible to fowl paralysis, due either to a special character inherited from the parent birds or due simply to the general quality of the stamina of the birds, and that the injection of the tissues from affected birds actually precipitated the development of fowl paralysis in the already receptive experimental birds to an extent of about nine times more than in the receptive control birds which were allowed to live normal lives without experimental treatment and which developed the disease to an extent which is commonly found among members of an infected flock in the field." Their present view is that fowl paralysis affects certain birds only—the progeny of susceptible fowls—and that although the tissues of affected birds contain some element or elements capable of producing accumulations of the typical types of cells, other conditions apart from the injection of such tissues may also precipitate infection.

(V) **Fowl Paralysis (Neuro-lymphomatosis Gallinarum).** J. BIELY, V. E. PALMER, and E. A. LLOYD, The University of British Columbia, Vancouver, Canada.

Field and experimental studies of fowl paralysis at this laboratory indicate that there is no connection between fowl paralysis and intestinal parasites. Furthermore, no direct connection could be established between the occurrence of fowl paralysis and any particular system of feeding or management. The feeding of yeast, lettuce, tomato juice, or colloidal iodine to birds affected with fowl paralysis has yielded no results. Recovery from fowl paralysis has been noted in several birds, but in such cases paralysis has re-appeared at a later age. Two birds showing paralysis of the right leg for over two years are still in excellent condition, and are laying. The earliest case of fowl paralysis in the experimental chicks was observed at thirty-seven days of age. Forty-five out of 248 chicks, or 18.4 per cent., developed paralysis before three months of age. Considerable variation was noted in the distribution and size of the lesions. However, lesions in the brachial plexus were more commonly found than lesions in the lumbo-sacral plexus, in spite of the fact that paralysis of the wing is less frequent than paralysis of the leg. About 20 per cent. of the females showed lymphomatous tumour of the ovary, and a smaller percentage showed the same type of tumour in other organs. Lymphomatous tumours of the testes were also observed in some paralysed males.

Clinical and post-mortem observation of birds affected with fowl paralysis reveals three conditions appearing singly or in various combinations: (1) paralysis;

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(2) lymphomatous tumours; (3) blindness. Since tissues other than those of the nervous system may be involved, fowl paralysis can no longer be regarded as a disease specifically affecting the nervous system. It should be considered rather as one of the manifestations of a specific malignant general disease with protean manifestations (lymphomatosis), in which any organ or tissue in any region of the body may become affected. Pathologically, fowl paralysis is characterised by a lymphoid infiltration or proliferation wherever the morbific agent or process occurs. Results of transmission experiments indicate that there are hereditary differences in resistance to fowl paralysis. The data show that resistance to the disease depends upon a dominant gene, and that in the stock used only one pair of genes is involved.

(B) THE CONTROL OF POULTRY DISEASES.

(I) **Control Measures of Poultry Diseases.** Professor BISANTI, Italy.

The author expresses the opinion that notifiable poultry diseases should include fowl cholera, fowl plague, fowl pox, fowl typhoid, and avian tuberculosis, and that regulations should be drawn up concerning the isolation of infected premises and the methods to be adopted for the rapid and complete stamping out of the disease in question. He thinks that immunising agents (vaccines, etc.) should be under State control, and that the use of live vaccines should be limited to poultry in already infected or adjacent areas. The movement of poultry from affected areas should be the subject of special legislation.

(II) **Preventive Measures against Infectious and Parasitic Diseases of Poultry.** H. MIESSNER, R. BERGE and R. WETZEL, Hanover, Germany.

The authors consider that the treatment of individual sick birds should not be considered and that only measures assuring rapid and efficacious results should be applied. They then deal with individual diseases.

Fowl Cholera and Fowl Plague.—These diseases are controlled by the veterinary police service in Germany. For fowl cholera, the simultaneous injections of serum and antigen is recommended.

Avian Tuberculosis.—The " wattle " tuberculin test has not given good results. The best method of control seems to be the renewing of the whole stock every one or two years.

Fowl Pox.—There is some difficulty in diagnosing the diphtheria type from catarrh and laryngo-tracheitis. Vaccination is recommended.

Fowl Enteritis.—This is important from the point of view of human food poisoning. Poisoning may result from the consumption of inadequately cooked infected goose liver and pigeon or ducks' eggs. The authors think that more strict control in the marketing of poultry should be exercised.

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Pullorum Disease.—This disease may be satisfactorily controlled by hygienic measures and the application of the agglutination test. It has been found that the rapid method with fresh blood is of equal value to the slow serum method.

Fowl Paralysis.—Recent research has shown that the disease characterised by nerve affections may be infectious, and that there may be some connection between the condition and leucosis.

Parasitic Conditions.—Blackhead may be controlled by sanitary measures. Tapeworm infestations are common. The intermediate hosts are snails, flies, beetles, etc., and the best measure in the control of the infestation is the elimination of the intermediate hosts.

(III) **The Most Important of the Poultry Diseases in France.** G. LESBOUYRIES, Alfort, France.

Professor Lesbouyries, Director of the station for the Study of Poultry Hygiene and Pathology at the National Veterinary School of Alfort, gives a table of the occurrence of the principal diseases of poultry in France. He describes the processes of protection and vaccination tried against these affections, and criticises, more particularly for bacillary white diarrhœa, the various methods used for detecting germ carriers (pullorination, slow or rapid sero-agglutination, and hæmo-agglutination). With regard to chickenpox, he indicates the satisfactory results obtained by preventive vaccination of the hen with pigeon virus and by the use of intramuscular injection of uroformine as a curative treatment. He reports the highly encouraging results obtained by feeding to young animals (birds and rabbits) affected with intestinal parasites (usually coccidiosis) thymolised cod-liver oil in the mash. The resistance of the animals is increased by this means, and mortality may be reduced and in many cases arrested. He describes the principal lesions of the most commonly occurring diseases of the hen, the pigeon and the rabbit, and indicates the various methods of treatment used in the control of these virus, microbial and parasitic diseases.

(IV) **Combating Poultry Diseases by the Government in the Netherlands.** B. J. C. TE HENNEPE, Rotterdam, Holland.

The poultry stock in the Netherlands is about twenty millions, and much is being done by the Government to combat disease. The treatment of fowls by laymen is not encouraged. The work of the Government can be divided into direct and indirect measures. The direct measures embraces the work of the Veterinary Service and the Government Serum Institute in Rotterdam, and the indirect measures apply to the training of veterinary surgeons in poultry disease work and the support given societies which make it their duty to combat disease. The Veterinary Service draws up and carries out measures regulating the import and export of poultry. In the Serum Institute a special department, under the direction of Dr. te Hennepe, has been set up to deal with poultry diseases. This

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department has five sections: (i) Post-mortem work and agglutination tests, all done free of charge; (ii) research into disease; (iii) preparation of vaccines and serums; (iv) statistical work; (v) instructional work on controlling disease, given to veterinary surgeons and poultry farmers. Veterinary surgeons are trained at Utrecht Veterinary School. As an example of the collaboration with poultry societies, the Netherlands Poultry Farmers' Society has appointed a Commission for combating B.W.D., and the Commission is subsidised by the Government.

(V) **The Legal Basis and the Organisation of the Campaign Against Poultry Diseases in Switzerland.** G. FLUCKIGER, Berne, Switzerland.

In Switzerland, fowl cholera, fowl plague and B.W.D. are notifiable diseases. The measures applied in their control have been followed by limiting their extent and occurrence. The author thinks that one of the essentials in controlling disease is harmonious collaboration between the police authorities, veterinary surgeons, and the poultry farmers. He further thinks that it is time now to consider the question of a campaign against contagious disease in connection with the export of birds, care being taken that any measures introduced are as simple as possible.

(VI) **The Extension and Eradication of Poultry Diseases in Denmark.** O. NIELSEN, Denmark.

Poultry keeping in Denmark is part of general farming, and the small flocks make it comparatively easy to control contagious diseases. Post-mortem work is done at the State Veterinary College. B.W.D. is being stamped out by the application of the agglutination test and by the control of the sale of baby chicks. Tuberculosis is being controlled by the application of the "wattle" tuberculin test—the State paying for this. There is a national breeding programme which not only deals with the types of birds used for breeding, but also deals with disease. Much of the success in the control of disease is due to this breeding programme as well as to educational work among poultry keepers.

(VII) **On the Infectious and Parasitic Diseases of Fowls in Sardinia.** G. PEGREFFI, Sassari, Italy.

Work during the past ten years indicates that fowl typhoid and fowl cholera have nearly disappeared following the use of polyvalent and stock vaccines. No B.W.D., fowl plague, or spirochætosis exists. Fowl pox can be controlled by the use of vaccines in the making of which phenol or formalin is used. Various types of paralysis exist, most of which appears to be due to parasitic infestation. Coccidiosis can be controlled by the use of whey acidified with acetic acid, together with the application of hygienic measures. The few cases of tuberculosis encountered appear to be of human origin, as shown by the results of the various tests applied to the bacilli isolated.

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Coccidiosis of a four weeks' old S. C. White Leghorn Chick.

(Reproduced from Kaupp's Poultry Diseases.)

First symptoms of muco-enteritis of a White Wyandotte hen. Note slight turning of head to one side, "drooped" wing, toes curled under the foot and slight reeling sidewise. Later all these symptoms become more aggravated, and finally the bird lies on its side unable to rise.

(Reproduced from Kaupp's Poultry Diseases.)

Head of cockerel affected with fowl-pox.

(Reproduced by kind permission from the Report of The Poultry Pathological Research Laboratory.)

Favus in a Mottled Ancona Cock. Circular cup-like scab formations are visible.

(Reproduced from Kaupp's Poultry Diseases.)

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RECENT PROGRESS IN THE INVESTIGATION OF POULTRY DISEASES.*

By T. DALLING, M.R.C.V.S.

Beckenham, Kent.

In deciding what to discuss at this meeting, the thought struck me that it may prove of value to review some of the disease conditions with which the poultry farmer has to deal to-day, and how research, especially research carried out recently or still being carried on, is helping us to understand to deal with them. Of course, it is impossible to deal with many, so I propose to select a few of the most important and confine my remarks to them. I think we might profitably discuss the present position of B.W.D., fowl pox, coccidiosis and fowl cholera, and then say a word or two on the general aspect of poultry disease.

B.W.D.

There is no need to reiterate the salient character of this disease. I shall refer to one point only, *viz.*, blood testing and its results. Despite what we read and hear, experience and results teach us that blood testing, with the removal of reacting birds from the breeding pen, is the only sure way of preventing this disease. It must be accepted that such a disease as B.W.D. exists, and that it is due to a specific microbe which is passed through the egg. (I do not propose at this stage to enter into arguments on the prevention of the disease by breeding from certain types of birds—that is another story which I shall touch on in connection with the general remarks I shall make on disease.) B.W.D. is a disease which became prevalent—the modern methods of breeding and keeping fowls may have been conducive to its rapid spread—and had to be dealt with. Research showed how it could be prevented, the blood test was popularised, with the result that much of the disease is being stamped out annually. Occasionaly we hear of failures in spite of blood testing, and wish to refer to some of the probable causes of such failures, especially those which have come to our notice.

(1) No test like the agglutination test can be 100 per cent. accurate, and no matter how carefully and accurately the testing is carried out, infected birds will very occasionally be missed because they fail to respond to the test. For example, there are birds which fluctuate in their response to the test, and may fail to react at a certain period when the test was made, while a test made at an earlier or a later date would probably give a positive reaction. In our experience, though such birds are uncommon, they do exist. Again, a truly affected bird may continually give what is referred to as a doubtful or a weak reaction; such reactions cannot be classified as truly positive, and some of them mean little or nothing (especially doubtful reactions), but because of the fact that some birds giving such reactions

* Read at Harper Adams Poultry Conference, August, 1933.

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are true " carriers " of the disease, all such reacting birds should be regarded as potentially affected and treated as true " carriers." Then the border-line reactor may give rise to a certain amount of confusion—a positive reaction may be got at one test, a doubtful or a weak reactor at another, and even a negative reaction at a further test. All these are types of birds which cause a certain amount of confusion in the minds of some poultry farmers, and even cause some to discredit the value of the test. Happily such types of reacting birds are rare. Our view is, and always has been, to regard every bird which reacts to the test, no matter in what degree and no matter how seldom, as a potential carrier, and discard it from the breeding pen.

(2) Non-reactors may become reactors and carriers of B.W.D. Examples of two methods of how this has occurred in our experiences may prove interesting and of value. After successfully cleaning up a breeding flock by repeated blood testing, we were puzzled and rather alarmed when we diagnosed true B.W.D. in some chicks bred from these birds. We were further concerned when on testing the birds again quite a fair percentage of the birds which had been passed negative on former occasions gave definitely positive reactions. After practically every avenue of possible source of infection had been explored, it transpired that some unhatched eggs had been fed to the breeding birds. Further inquiry produced evidence that the eggs which were fed had not been produced by the birds in question, but had been purchased from an outside source. From the losses reported among chicks from the same source it could be concluded that B.W.D. was present in the eggs. There, then, is one source of infection of adult stock. The other method of which we have had experience is the transmission of the actual B.W.D. microbe from " carrier " reacting birds to clean fowls. We have carried out quite a lengthy investigation into this point, and have demonstrated very clearly that such transmission can occur, and is dependent largely on the number of reacting and non-reacting birds kept in the same pen, and also on the size of the pens.

(3) It must also be borne in mind that the B.W.D. blood test is not specific, but that it detects birds affected with or " carrying " infections other than B.W.D., the most important of which is fowl typhoid and *B. gaertner* infections. Fowl typhoid can be transmitted from parent to chick in a manner exactly similar to B.W.D., and young chicks may die from fowl typhoid presenting symptoms very much akin to B.W.D. Further, unless careful post-mortem work is carried out, the two diseases in the young chicks are difficult to differentiate. There is also some record of *B. gaertner* infection behaving in a similar way. It stands to reason, therefore, that should fowl typhoid (which appears to be prevalent in some parts of the country) or *B. gaertner* infection make its appearance in a breeding flock of negative reacting birds, the damage done may be erroneously ascribed to the unworthiness of the B.W.D. agglutination test.

Our advice, gained by experiment and experience, is to guard a B.W.D. clean breeding flock jealously—prevent any chance of its becoming infected, for it is

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probable that a clean flock, especially one that has been clean for some time, becomes susceptible to introduced infection. Before leaving the subject of B.W.D. I should like to point out that although B.W.D. in its acute form is essentially a disease of young chicks, outbreaks may occur among adult stock, especially during their first laying season. Our experiences indicate that losses from this cause are becoming more common. Blood testing is again called for in connection with such an epidemic. Lastly, we read much of the various methods advocated for the carrying out of the blood test. We have experience of all of them, and find all useful in their own way; in fact we have to resort to different methods under different conditions, but we still find that for convenience the so-called tube or long method is the best, and gives highly reliable and satisfactory results.

Fowl Pox.

Fowl pox is an example of a disease the occurrence of which the results of recent research have been able to reduce very considerably. I refer to the results of vaccination. This method of prevention is now in common use, and where properly carried out excellent results follow its application. There are several points in connection with the vaccination method, all of which are of the greatest importance in the obtaining of satisfactory results: (1) The vaccine must be effective. The commonly used vaccine is, as you know, the living virus contained in the scabs produced by pigeons when infected with pigeon-pox. The virus contained in the vaccine must be alive, and must be capable of producing a reaction when applied to the scratched surface of the feather follicles of the vaccinated fowls. This is the problem of the laboratory worker producing the vaccine—he must be satisfied that the vaccine issued is alive, and will produce such a reaction. When the ground-up pigeon scabs containing the virus are mixed with the glycerine to form the vaccine, there is a tendency for the virus to die within a short period, hence most laboratories issuing fowl pox virus vaccine to-day stipulate that it must be used within a certain time. The experience of the laboratory worker is behind such advice. As perhaps you know, actual fowl pox virus is largely used in America for preventive vacination against fowl pox. Our experience with such a vaccine shows that in this country at least a certain amount of danger is attached to it, and we refrain from recommending its use. On the other hand, we find that if such a fowl pox virus be weakened somewhat a highly efficient and satisfactory vaccine may be produced. It is probable that the results of further work will cause such a vaccine to be largely used in this country. (2) The vaccine must be properly applied. Many poultry farmers have acquired the " knack " of vaccinating birds. It is a procedure which can be acquired with practice, and with care, and must be properly carried out for the production of good results. There are still some farmers, however, who have not acquired the technique necessary for vaccinating their birds. It must be remembered that the method consists in applying a sufficient quantity of the vaccine to a raw area produced by removing the feathers (six to eight), and lightly scratching

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the skin so as to cause a certain amount of injury to the top layers of the skin without causing bleeding to any extent. We find that errors arise because the scratching is done too hurriedly, so that the skin is either not injured sufficiently deeply or the injury has gone right through the skin, and much bleeding has occurred. Properly applied vaccine should cause some degree of reaction (swelling of feather follicles with production of scabs later) within about a week, and unless this reaction is produced the vaccination has not been properly carried out, and in all probability the birds will still be susceptible to fowl pox. (An exception to this is the bird which is already immune by having passed through a fowl pox epidemic.) This reaction should *always* be looked for, so that the farmer will know whether or not his birds have been properly vaccinated. (3) Healthy birds *only* should be vaccinated. Investigations into the few outbreaks of fowl pox reported to have occurred soon after vaccination has been carried out have shown that in some cases at least the birds were actually in an affected state when the vaccination was carried out. It must be remembered that birds may be affected with fowl pox without the various symptoms being evident, and that the mere scratching of the skin is sufficient to cause the usual pox to develop. (4) When vaccinated birds develop a disease resembling fowl pox it should be ascertained whether the disease present is really fowl pox. Several diseases bear a certain resemblance to fowl pox, and on first sight some confusion with true fowl pox might exist. In cases of doubt, laboratory assistance should be sought, for if necessary an actual transmission experiment may be performed for confirmation. Birds developing fowl pox some months after vaccination have been reported and examined on several occasions. As a rule it is found that such birds have certainly developed some symptoms and lesions of true fowl pox, but that the disease is strictly limited in its character and extent; in other words, the immunity produced by vaccination has gradually weakened, but is still sufficient to limit the infection. Such birds rapidly recover, and are then permanently immune.

There is one last point I should like to make. In the treatment of any animal, including poultry, for the production of an artificial immunity to a disease, the general conditions under which the animals are kept play a high part in the production and maintaining of that immunity. If the animals' system is taxed to its greatest extent in warding off other diseases, and in keeping the general body in a more or less healthy state, the extra response necessary for the production of the specific immunity may not be forthcoming, and even the immunity which has been acquired may be rapidly lost. From such a statement you will gather that although vaccination against fowl pox has been carried out efficiently, the poultry keeper must still pay attention to the general conditions under which the birds are kept to ensure the best results.

Coccidiosis.

I do not intend to go deeply into the subject of coccidiosis, because the cause of the disease is well known, and there are several methods recommended by

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various authorities for the prevention and cure of the disease, all of which methods have their strong supporters. Much work on coccidiosis, both as affecting the young chick and the growing bird, has been done in America, and we in this country are now concentrating on some of the findings of the American workers. We must recognise that the disease does affect young chicks, and causes losses among them. It was only during the last year or two, however, that attention was drawn to what is called the duodenal form of the disease which affects older birds. In the minds of some people there exists some considerable doubt on the question of the occurrence of epidemics of coccidiosis among these older birds. It would appear that coccidia are found in many birds which are apparently healthy, and in which no recognisable damage can be found from their presence in the intestine. On the other hand, it is by no means uncommon to carry out post-mortem work on a young bird and find the intestine simply teeming with coccidia which have apparently given rise to gross changes in the wall of the gut causing the bird's death. Our experience in this matter is that in some so-called epidemics only some birds in a flock are so affected, and that it has been wrongly deduced that other birds in the flock are likewise infected. The impression I wish to convey is that in all probability coccidia in a healthy intestine do practically no damage at all even when considerable numbers are found ; trouble arises, however, if the bird is in a condition of susceptibility which allows the natural resistance of the intestinal wall to be overcome. Such conditions are probably many, not the least important of which are the natural stamina of the bird and the methods adopted in the rearing and feeding of the bird during its early life and growing stage. We have noted on many occasions that coccidiosis has given rise to trouble in a certain percentage of one group of birds only on a farm. In some cases all the groups of birds had been actually hatched on the premises and reared on the same food and by the same method, though they had been bred from different stocks ; while in other cases the birds had been bred from the same stocks, but reared under different conditions and fed on different food materials.

Our experiences indicate, therefore, that in the adult or duodenal form of coccidiosis there is something more involved than the mere " infection " with coccidia, and that probably methods of breeding, rearing and feeding play the more important part in the production of coccidiosis. (I shall refer to this in a later part of the paper.)

In connection with coccidiosis in general, it may interest poultry farmers to know that chicks are hatched free from coccidia. We have no evidence that the disease is passed through the egg, or that the cysts which give rise to the disease are harboured on the egg shell. It was thought at one time that cysts could remain alive on the egg shell and infect chicks on hatching, but experiment and observation have failed to prove this point. We have repeatedly kept chicks obtained as day-old birds from various sources for several weeks, examining the droppings almost daily ; these birds have always remained free from coccidiosis. It has also been shown that one single cyst is capable of infecting a susceptible

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chick. As a rule this cyst develops in the intestine, causing a small amount of damage ; the various stages of further development are gone through, and a multitude of new cysts are produced which pass out in the droppings. You will gather from this that one single cyst does not do a tremendous amount of damage, in fact our experience is that probably several thousand cysts are necessary to cause sufficient damage to kill the bird. In our opinion the great damage in coccidiosis is caused by repeated infestation. The first lot of cysts may cause a certain amount of damage, thus laying the wall of the intestine open to attack by further infection of cysts as they are picked up. As you know, the cyst when it passes to the outside must undergo a change called " sporulation " before it is in a condition capable of causing further infection when swallowed. The time taken for this change to take place varies enormously with the weather conditions. We have shown that given the best possible conditions, the shortest time in which such sporulation can occur is about twenty-four hours. Under ordinary weather conditions it is possible that this period is considerably longer. It follows, then, that the methods to be adopted for the prevention of coccidiosis in susceptible birds include the removal of cysts from the intestine of birds before they can do much damage, the rendering of the intestine into a state which will prevent further changes taking place in the coccidial cysts, and the prevention of re-infestation either by actually destroying the cysts passed out in the droppings of infected birds, by rendering them incapable of sporulating, or by removing the birds from the chances of picking up sporulated cysts.

Fowl Cholera.

Within recent days we have read and heard a lot about an epidemic of fowl cholera in this country. Precise information on the subject has been very difficult to acquire, but it is stated that the disease originally affected young ducks, and reports indicate that numerous birds have been lost. It is further reported that it seems probable that the disease was imported into this country. It is also commonly stated that fowl cholera has not existed in this country for some years. I think, however, that our investigations over a period of years permit us to refute this statement, for we have records of about a dozen outbreaks in different parts of the country. It is true that these outbreaks, with a few exceptions, did not assume large proportions, but we have no hesitation in saying that in all these cases to which we refer we have demonstrated by the recognised tests that the germ which causes fowl cholera was present in birds which had died or which had been killed. Our first experience of the disease was in March, 1931 ; we reported this and other outbreaks in the press in January, 1932. While we willingly accept that fowl cholera as an acute affection in which large numbers of birds have been lost has not been seen in this country for some time till recently, our opinion is that in some flocks fowl cholera exists in the form of a chronic disease which birds may harbour without showing any marked symptoms of illness. When some untoward circumstance causes these birds to become lowered in resistance to disease they themselves may fall victims to fowl cholera, and may

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in that state spread the disease to other birds. There seems to be little likelihood of the disease affecting a large number of birds by contact with a chronically affected bird, provided the birds have no intercurrent infection and provided that they are living normal existences. A period of very hot weather would appear to be conducive to a spread of the disease. We pointed out in the press some time ago that cockerels suffering from a swollen condition of the wattle very often harboured the germs of fowl cholera in the affected wattles. We have seen this condition on several occasions, and though the birds appeared to be perfectly healthy, still the germs were present, and were capable of setting up infection when introduced into the body system of other healthy birds. Another condition in which we have found the fowl cholera germ is in one of the types of so-called catarrh. An examination of discharge from the nostrils of birds in certain flocks has revealed the presence of the typical germ. The fowl cholera germ seems to vary tremendously in its capability of producing disease. Some varieties would appear to be incapable of causing much harm, while others are very virulent, and cause symptoms of true fowl cholera when conditions in the bird are suitable for their multiplication. We have also met with the disease in young ducks.

It is difficult to know how to advise the clearing up of fowl cholera in a flock. If the disease assumes acute proportions, as it apparently has done in these recent outbreaks, the further prevention of the spread of the disease is fairly simple. Prevention of traffic in birds from the affected farms, ruthless killing, and disinfection and isolation of premises are certainly called for. Where the disease exists in a chronic form, and where only an occasional bird dies or a small group of birds die, the problem of ridding the flock of the infection becomes more difficult. It seems possible that under such circumstances suitably prepared vaccines may have the effect of raising the resistance powers of the birds to such a degree that they will withstand infection, if the disease tended to assume the acute form. Vaccines have not yet been tried in this country to any extent, but it seems to us that there is a possibility that their use would be followed by beneficial results.

General Remarks on Disease.

In reading *Feathered World* the other day I came across an article by Sir Edward Broom entitled "A Noteworthy Book." In his review he wrote the following: "It is a somewhat surprising thing that disease has become much more serious in almost every country correlative with study of such disease on the part of specialists. I do not suggest that one is the result of the other, but that at the time and subsequently when scientists began to study poultry diseases there was introduced, in order to augment production upon specialised lines, methods of breeding, feeding and management which have seriously affected the stamina of the stock. They are susceptible to an extent not previously known."

In the same article, commenting on a statement that there is a predisposition of certain breeds to various forms of disease, Sir Edward says: "If that be so, may it not be that the diseases which are present at the present time in various

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directions are the result of bad methods of both breeding and production, and that the poultry-keepers as yet are more concerned with curing the birds, or endeavouring to save their lives, than to preventing any tendencies they may have to be communicated to progeny ? "

I do not think that I could put more clearly the views concerning disease in general which I hold. I am convinced that much of the disease in birds in this country, diseases of any infectious nature which I have been discussing, as well as the " vague " type of disease with which one cannot associate any specific cause, are coincidental with lack of stamina, which in its turn is coincidental with " artificialdom." Leaving aside for the moment the diseases of an infectious nature, as well as those of a non-infectious type, but which we can diagnose with a name, every worker who has carried out many post-mortem examinations must be struck with the large number of birds presenting literally no lesions, or lesions which one hesitates to describe as the whole cause of the bird's death. In my opinion, such birds have as the root cause of their trouble simply lack of stamina. By stamina I think we infer the power to lead the life demanded of them and resistance to disease. I do not think there is a more fitting example of lack of stamina than worm infestation. A very heavy infestation with worms—especially tapeworms, the nature of which we know, thanks to the work of Dr. Bayon—may cause some loss in birds, but unless the subjects lack stamina, or are suffering from some undercurrent infection, I suggest that there is little evidence to show that they are disease-producing. I hold, rather, that the presence of worms should lead one to search further for the root cause of losses in a flock, and I predict that in many cases the trouble will boil down to lack of stamina. I venture also the opinion that in some cases at least infestation of growing birds with coccidia comes into the same category. In the field of true infectious disease stamina also plays its part. I do not for a moment suggest that birds whose stamina is of the desired degree will inevitably pass through an outbreak of infectious disease untouched, but I do hold that the numbers affected and the intensity of the infection will be materially influenced thereby. What causes lack of stamina ? Broadly speaking we can sum up the cause in one word—" artificialdom "—and the degree of each is closely inter-related. Further, again speaking generally, " artificialdom " and resultant lack of stamina is found in breeding, general management and feeding. It is not uncommon to find a single group of birds on a farm affected with a parasite infestation or some other disease condition causing deaths, while other groups remain free. Inquiries show that all the groups of birds on the farm have been reared, housed and fed in an exactly similar manner, and have been submitted to the same general management conditions ; the difference is that the infected groups were obtained as eggs, or as day-old chicks from a special source. We deduce from such experiences that the lack of stamina in the group is attributable to breeding errors. And as one considers the haphazard way in which some breeding is carried out, expecting birds to be able to reproduce under every or any conditions, the transmission of

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lack of stamina does not cause much surprise. Again, there is the case of birds all from the same original stock, but divided into groups and subjected to different conditions of rearing: certain groups only show evidence of lack of stamina by their falling victims to disease when submitted to equal chances of infection as other groups reared under different conditions and which prove practically resistant. Lastly, there are the groups fed on different types of food and subjected to different methods of feeding. It is no uncommon occurrence to find marked lack of stamina in certain groups only, though all originally came from the same parent birds. Lack of stamina may be in no way evident by the general appearance of the bird nor by its bodily condition. Exposure to disease of any nature will soon give the necessary information on the subject. The period just prior to the beginning of laying is the critical time—an extra strain is put upon the bird's system at that time, and stamina will tell its own tale then.

I do feel that it should be very clearly realised that if the highly artificial methods under which poultry are bred, reared, fed and managed, are necessary from an economic standpoint, then disease and more disease will inevitably follow. I have a strong feeling that the average poultry keeper is not fully alive to this fact, and that he fails to realise that he himself is responsible for much of the disease in his birds to-day. He cannot prevent epidemics entirely, nor the odd case of disease of a " vague " nature among his young birds, but by adopting reasonable methods of breeding, rearing and general management he can assist himself in preventing disease, because his stock will be in a better condition to resist the occurrence of such disease conditions. The resistance to disease in general must be a problem for all branches of the poultry industry, including the poultry-keepers, the breeders, the feeding experts, the general management advisers, etc., and the disease workers. I am firmly of the opinion that though disease workers may be able to help the poultry farmer to a considerable extent in curtailing his epidemics, the problem of disease will never be satisfactorily tackled till the various branches of the industry get together and work together: each and every one is intimately concerned with the future of disease, bound up as it is with stamina.

I finish this address with an extract from another article, written this time by Mr. Tom Newman in a recent issue of *Eggs*: " We must recognise that the constitutional vigour of the bird is of primary importance, and how that may be impaired during rearing is a question to which the industry and all connected with it should devote their most careful consideration. One is concerned to see, in all laying tests, the number of birds which are now dying from what is described as *egg peritonitis*. Is this a matter of breeding, or is it a question of feeding? In my opinion it is primarily a matter of breeding; secondly, a question of rearing." And in the same article is found: " Poor condition is not always a matter of nutrition, in fact we may safely say that in the vast majority of cases it is not a question of nutrition, but rather one of environment or heredity: a weakened constitution or conditions of life which prevent the proper assimilation of the foods supplied."

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POULTRY BREEDING RESEARCH IN BRITAIN, 1930-1933.*

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In no country in the world has the keeping of poultry, whether for sport, fancy or profit, attracted so many adherents as in Britain. The number of organisations devoted to promoting the interests of particular breeds runs into hundreds. Yet in spite of this long-standing and widely-spread public interest in poultry breeding, very little public provision is made in this country for research work into the problems of heredity in poultry. This no doubt comes from the national prejudice in favour of empiricism and the Englishman's characteristic suspicion of pure knowledge. Be that as it may, the practical consequence is that in the three years since the last World's Poultry Congress no great amount of research work on poultry breeding has been published in Britain.

By far the most interesting discovery to be announced since the last Congress is that of Professor Punnett relating to sex-linkage in ducks. The wild Mallard has a dark down ; the Fawn Runner duck (and some other breeds) has a lighter one. This difference in down colour is due to a sex-linked factor, the darker being dominant to the paler down. In other words, when a pale-coloured fawn Runner drake is crossed with dark Mallard ducks, the cross-bred drakes have dark down and the ducks pale down, as is shown in the British exhibit. This is the first case of sex-linkage in ducks to be put on record, and the discovery is clearly one likely to be of considerable use to the breeders of Runner ducks, who find it hard to dispose of the surplus drakes. Professor Punnett is engaged in further work on sex-linkage and fecundity in poultry and on the inheritance of shell-colour in eggs, and his future results are awaited with interest by all.

Last year Dr. A. W. Greenwood, of Edinburgh University, published an interesting paper on the value of progeny in relation to age of dam. It is generally held to be bad practice to breed from pullets ; yearlings and older birds are considered to give better progeny ; indeed, Professor Pearl has gone so far as to predict that a race of super hens could be produced by breeding only from very old hens. Dr. Greenwood has, however, published some carefully analysed data which show that as regards fertility, hatchability and egg production there is nothing much to choose between pullets and yearlings ; but that in subsequent years there was a very large falling off in these characters. For example, in the seventh year the hatchability was only 22 per cent. of that in the first year. It would be interesting to know if the decline in hatchability was due to changes in the quality of the shell, or in the constitution of the embryo ; it is to be hoped that Dr. Greenwood's final results will throw light on this and the many other interesting lines of thought to which his preliminary figures give rise.

* Read at World's Poultry Congress, Rome, September, 1933.

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Ever since sex-linkage came into the poultry industry, poultrymen have heard talk about chromosomes. But the actual chromosomes in the chick cell are very difficult to see. They are very small, there are very many of them, and they tend to clump together during the process of killing and staining the material for the microscope. Only this year has a satisfactory account of the chromosomes in the chicken appeared. Mr. White, of London University, has, by means of a greatly improved technique in fixing, shown that the fowl has 66 chromosomes in the male and 65 in the female. The chromosomes which are presumed to be the sex chromosomes are very much longer than the others. The whole cytological picture accords very well with what is known of poultry genetics, namely, that there are several sex-linked factors and many other independent (*i.e.*, not linked) factors in the fowl.

An interesting note by Mrs. Bisbee, of Liverpool University, has appeared describing a semi-featherless condition in Rhode Island Red hens, almost certainly due to inherited factors. Since every degree of this abnormal condition appears in progeny from such stock mated to normal birds, it is clear that the matter is complicated ; Mrs. Bisbee's fuller account is awaited with interest.

At Reaseheath, in the North of England, there is a station for poultry breeding directly supported by a Government grant. Work here has been devoted to problems of in-breeding as they affect the practical poultry man. A preliminary report of the work was made at the last World's Poultry Congress ; further work has gone to confirm that in White Wyandottes, at any rate, in-bred stock are inferior to out-bred stock by about 25 per cent. in rearability and in fecundity. This figure may well vary from strain to strain and from breed to breed. But in general there is very little doubt that in-breeding in utility stock very quickly brings about a rapid deterioration in the utility qualities. The discovery of a strain of poultry capable of resisting the strain of close continuous in-breeding is an event for which both science and the industry eagerly awaits, and for this (as in most else where poultry science is concerned) our eyes turn to the West.

I now come to work for which I have been personally responsible, and if I deal with this at some length it is not in the least because I regard it as more important than other work, but because of it alone can I write with first-hand knowledge. Moreover, most of what follows here has not been published elsewhere. At the last Congress in London there was shown in the British Exhibit a new race of poultry (called the Cambar) which breeds true and shows sex-linkage within the breed. As a full account of the genetics of this race was published at the time of the last Congress, nothing further need be said now about that aspect of the Cambar. Clearly this character of sex-linkage within a pure race is one of considerable economic importance. But unfortunately the Cambar as originally produced, though attractive to look at, had no utility character to recommend it ; its eggs failed in number, in size, and in colour. To turn the Cambar into a utility breed is a nice problem in applied genetics.

The work started with twelve pullets and two cocks. Of the pullets two died

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without laying any eggs ; one laid 162 eggs in her pullet year, but not one of these eggs was fertile. The remaining birds scored from 114 eggs down to 41 in their pullet year, winter production being practically *nil*. As for egg weight, only one bird laid eggs of a reasonable size (62.9 grammes) ; the remainder ranged from 58 to 49 grammes. In colour the eggs varied from white to cream. Since these birds had been produced by close in-breeding, without any particular selection for stamina and hatchability, there was every reason to believe that to multiply the stock by direct in-breeding would be difficult. This indeed proved to be the case, since in 1931 only six pullets and five cocks were raised to maturity by direct in-breeding, and in 1932 none survived adolescence. However, since the genetic make-up of the Cambar pattern was exactly known there was no inherent objection to cross-breeding. The Cambar cock should in theory, when mated to Campines (gold or silver), give pure Gold Cambar pullets. The trouble, however, is that in Britain at any rate the Campine is not a utility variety. However, two such Campines which at least showed some sign of winter production were mated to one of the original Cambar cocks. As expected, all the twenty-two pullets from this mating were pure Gold Cambars ; unlike their in-bred half-sisters, they were easy to rear, even though late hatched. Their winter records were not good ; but all things considered, the average of 153 for the pullet year from the unculled pen was not an unsatisfactory start.

To improve the winter records was clearly the most important matter ; for this purpose some Belgian Campines from the " Station Officielle de Sélection pour la Race Campine " were imported. Some of these birds had competed in the Belgian national egg-laying trials (Nationale Legwidstrijd) at Rousselare, and had finished second in 1931 with tolerably good scores of about 200 eggs each. These birds, mated to the best two of the 1931 gold cockerels, gave 150 Gold Cambar pullets, which are now being trap-nested. At the time of writing it is not possible to say anything about the full year's records of these birds ; but as far as winter records go, they show a great improvement on their fore-runners, averaging 41 eggs per bird for the winter months (October 1st to February 28th) as against 27 eggs per bird for the unimproved Cambars trapped side by side over the same period. There was considerable variability in egg size, but of the 150 pullets, 33 have given eggs consistently over 60 grammes in weight. (It may be remarked in passing that 12 of these 33 birds came from one hen who herself laid big eggs.)

It seems clear, then, that there is not likely to be great difficulty in levelling up the Cambar to the Campine standard. Ordinary selection should get rid of the shy winter layers and the producers of small eggs. But it is doubtful if selection will lead the breed beyond the Campine standard, and this standard falls woefully behind that of the industry of to-day.

Some other outcross, therefore, has to be resorted to to bring into the Cambar the utility qualities which it lacks, and which the trade of to-day requires—brown eggs, large size, and consistent winter egg production. In choosing an outcross, a consideration of the genetic make-up of the Cambar down pattern played the

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principal part. It is important to be able to extract from such a cross the wanted pattern sharply defined at hatching, in as large numbers as possible, and with the certain foreknowledge that as extracted the birds will breed true. The Barred Rock provided the best chance of fulfilling these conditions, and to the American continent we turned for high-grade utility Rocks. The University of British Columbia provided a trio of Barred Rocks with an impeccable pedigree of utility performance. The first-crosses from these birds were not unlike Rocks in plumage, though slighter in build and more handsome in carriage than the pure breed. The pullets laid well, size and colour of egg leaving nothing to be desired. From these birds by direct in-breeding 760 chicks were hatched, of which nearly a quarter (170) showed Cambar down or something near it. Of these chicks, however, only 39 pullets survived the accidents of rearing and the scrutiny of culling for correct pattern, and these are now being trapped. The back-cross (half-bred male to pure Cambar pullets) gave 240 chicks, of which rather more than half had Cambar down or something approaching that pattern. After culling for correct pattern, 43 pullets survived to go into the trap-nests.

At the time of writing, any detailed account of the year's performances of these birds is not possible. From the nature of the mating, we are not in this case concerned with any average figure. We are looking for the one or two individual Cambars which may have come out of this cross carrying with them the utility characters of the Rock. Certain it is that amongst the extracted Cambars there are several which have got both the size and colour of egg from their Rock grandparents. As regards fecundity, the only figures so far available are those of winter production, not, however, a bad index. It may perhaps be regarded as hopeful that six of these pullets laid more than 50 eggs in this period. But much more work will be required to fix these elusive utility characters in the Cambar. Progress is bound to be slow, since for egg characters the male can only be judged indirectly by his progeny.

Two interesting Mendelian bye-products emerged from the Rock-Cambar cross. The original Cambar was gold and hen-feathered like its ancestor the Gold Campine. Both these characters are determined by simple Mendelian genes, of which the Rock carries the alternative genes, silver and cock-feathering. Since the Rock was the male parent, the crossbreds were silver and hen-feathered. In the next generation there appeared, by the process of ordinary Mendelian shuffling, the Silver Cambar and the Cock-Feathered Cambar. The Silver Cambar shows the characteristic sex-linkage difference in the downs; if anything the difference is more striking than in the original golds. The Silver Cambar has the advantage over the original Gold Cambar that the hen can be used for ordinary sex-linkage both with respect to the Silver-Gold character pair (*e.g.*, with a Buff Leghorn) or with the Barred-Black character pair (*e.g.*, with a Black Leghorn), both of which crosses are shown in the British exhibit.

Two minor applications of Mendelism may be mentioned. The Silver Cambar cocks must be tested for purity of silver; this has been done, and several families

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of pure Silver Cambars now exist. The other problem is this: Since hen-feathering is dominant to cock-feathering, all the cock-feathered cocks will breed true. But what of the hens? Except by progeny testing, there is no way of knowing which of the Cambar hens are carrying the gene for hen-feathering, and which do not, and therefore will always breed true for cock-feathering. A pure strain of hen-feathered Silver Cambars can be more conveniently made by extracting from the Silver Campine × Cambar cross. Indeed, the hen-feathered Silver Cambar cock shown in the British Exhibit has been made in this way. As far as utility is concerned, there is no reason to suppose that the type of feathering of the cocks makes any difference one way or the other. But since from the Rock cross cock-feathering is more easily fixed, this will probably be adopted as the standard of the breed. This preliminary account of the development of the Cambar raises many points of scientific and practical interest, and it is hoped that as the work proceeds much useful information will be collected and become available to all interested in the advancement of poultry.

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THE PATHOLOGY OF FOWL PARALYSIS, WITH SOME ASPECTS OF ITS CAUSE AND CONTROL.*

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Introduction.

Fowl paralysis was first described in Europe by Marek (1907), and later in America by a number of observers, including Kaupp (1921) and Pappenheimer (1926). It was given the name of "neurolymphomatosis gallinarum," presumably owing to the supposed constant presence of lymphoid cellular swellings of nerves that were observed by pathologists. In America it has acquired the name of "range paralysis," possibly to distinguish it from the paretic condition seen in polyneuritis arising from avitaminosis, which may occur in birds that are restricted to a deficient diet. There always has been a decided difference of opinion as to the cause of the disease, and there still seems to be a strong leaning

* Paper read at the Harper Adams Conference on August 16th, 1933.

towards the belief that coccidia or other intestinal parasites are the direct cause, with a certain section of observers.

Galloway (1929) described the condition in England, and Warrack and Dalling (1932) published the results of valuable investigations, but notwithstanding the sound work that has been done on the condition, few diseases have lent themselves to such varied and imaginative speculation. Actually, from a considerable amount of evidence accumulated, the condition appears to be caused by a virus, which is neurotropic, but not exclusively so, because besides infiltrating the brain, cord and peripheral nerves, it produces in roughly about 10 per cent. of cases cellular aggregations in the organs and other tissues of the body. These cell growths resemble some other tumours rather closely, and this may give rise to a mistaken diagnosis, particularly in some forms of the leucosis that affects fowls.

Relative to some superficial points of confusion between fowl paralysis and the leucoses may be mentioned the findings of Patterson, Wilcke, Murray and Henderson (1932). These workers consider fowl paralysis and the leucoses as manifestations of a multiple pathology derived from a unit ætiology. This has been kept carefully in mind during the investigation, but a detailed microscopic study of the blood and bone marrow in many scores of cases of fowl paralysis demonstrated clearly the lack of erythroblastic or myeloblastic hyperactivity, as well as the total absence of many other features which are essentially present in erythroleucosis and myeloid leucosis.

In the inter-connected conditions of erythroleucosis and myeloid leucosis, a cellular excess of immature red cells in the former and of immature granular white cells in the latter are found in the blood stream combined with an aggregation of these cells in various organs of the body, producing at times tumour-like growths. These conditions are transmissible, and are probably caused by a virus. An allied condition known as lymphatic leucosis, in which the blood cells are not altered, but in which there are lymphocytomatous growths in the liver and other organs, is usually held to be non-transmissible. The leucoses in some aspects lie rather close to true cancerous growths, particularly to some of the more primitive cellular types that are found among the sarcomata.

Several of these types of sarcomata observed in fowls are easily transmissible, but this has not yet been found to be the case with the rather more specialised cancers or carcinomata, some of which also are seen in fowls. Fowl paralysis in some of its clinical and histo-pathological manifestations shows points of resemblance to other neurological conditions, such as acute anterior poliomyelitis or infantile paralysis. In both conditions microscopically there is found a somewhat similar infiltration of the nervous system, with lymphoid cellular hyperplasia and nerve-cell degeneration. Poliomyelitis belongs to the group of diseases, of which rabies and borna disease are members, in which the virus travels along the nerve filaments, and in so doing provokes a lymphocytic infiltration of the nerve which is of the nature of a cellular reaction to it. In this they strike a

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note of resemblance to fowl paralysis, but there are even closer features than this in the case of borna disease. This condition affects horses, rabbits and other mammals, but fowls are susceptible, and it was transmitted to them by Zwick, Seifried and Witte (1926). Pathologically, borna disease consists of an encephalomyelitis with a universal neuritis, particularly affecting the autonomic ganglia and nerves, and consequently producing symptoms referable to any organ innervated by these nerves.

It will later be shown that these are all salient features of fowl paralysis, and, additionally, it may be noted that the virus of borna disease frequently shows a prolonged latency in susceptible animals, which is a trait that fowl paralysis exhibits on occasions. A distinctive aspect in fowl paralysis arises in the cellular hyperplasias sometimes found in various tissues, but it has been pointed out by McGowan (1927) that these can be provoked in the fowl by a variety of tissue irritants—mechanical or toxic. Regarding this group of neurological conditions, it is of interest to note that McIntosh and Scarff (1928) pointed out the resemblance histologically between infantile paralysis and borna disease, and Nicolau and Galloway (1928) were able to obtain cross immunity to a certain degree between the two diseases.

Clinical Diagnosis of Symptoms.

It would appear after each breeding season since 1930 that fowl paralysis has become more widespread in this country, until it is no exaggeration to say that it has decimated many flocks, and is a calamity to all sections of the poultry industry. At the outset the condition is not so easy of correct diagnosis, because almost any severe disease, especially abdominal conditions, will send a bird off its feet through muscular weakness, as occurs with rickets, avitaminosis, coccidiosis, intestinal parasitosis, or any other condition producing general weakness, including the leucoses and cancerous tumours in their later stages.

Even drooping of the wings may be met with in conditions producing muscular weakness, but it does not proceed to the extent of a complete paralysis. In fowl paralysis the paresis of the legs varies according to the stage of the disease or the type of case. Sometimes a completely flaccid or limp paralysis is seen, but when there is degeneration proceeding in the upper motor neurone, or there is irritation of the lower motor neurone or motor nerves, spastic paralysis occurs, with contracture of the limb muscles and the digits. Loss of muscular control occurs, especially in the weaker extensor muscles, while the flexor muscles tend to overact. This is demonstrated in the jerky gait seen in some early cases, whereas in the flaccid, limp type, the bird tends to drag the leg. The reflexes are in some cases exaggerated, depending on the lesion in the cord and peripheral nerves.

In muscular weakness, such as is met with in rickets and coccidiosis, the reflexes are not lost, but in true fowl paralysis there is eventually complete loss of the extension and fanning of the digits seen normally when a bird is suddenly lowered towards the ground. The ultimate end of muscular paralysis is atrophic wasting of the muscles, which often becomes very marked.

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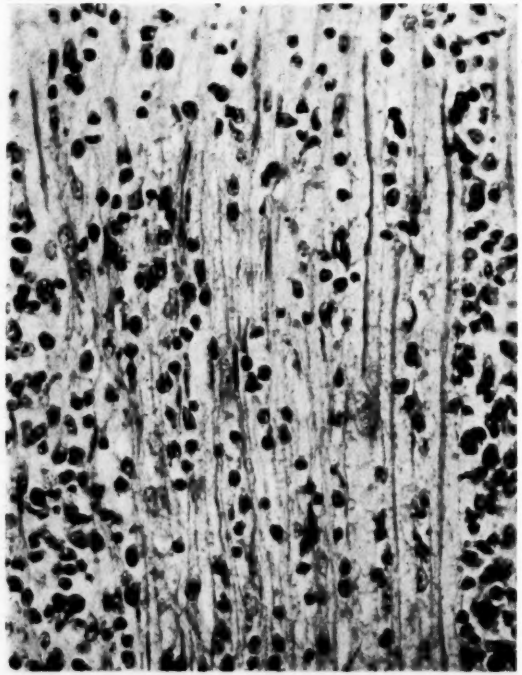

FIG. 1.—Cellular infiltration of lymphoid and plasma cells in nerve. (×450.)

FIG. 2.—Lymphoid cells infiltrating the muscle bundles of the heart. (×350.)

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When the brain is involved the bird is often restless and easily excitable, or, on the other hand, it may be lethargic. Some of these cases develop a spastic condition of the neck muscles with weaving or burying movements of the head, due apparently to involvement of the spinal accessory, or eleventh cranial nerve. In some of these cases the onset of severe symptoms may be very sudden and striking. After showing mild symptoms a bird may be a sprawling, twisting heap inside a very few hours. Types of fowl paralysis with transient slight symptoms, usually lameness, frequently occur, from which the bird sometimes recovers.

Arrest of the disease is also seen in some of the cases which show depigmentation of the iris alone or combined with irregular constriction of the pupil. Many of these iritis cases escape detection in flocks, and are a great potential source of infection. When other parts of the sympathetic nervous system are involved alone, diagnosis clinically becomes difficult, because it is possible to get any one of the conditions of crop-binding, stasis of the proventriculus, gizzard, intestine or cloaca, due to interference with the sympathetic nerve, innervation of the muscular coats and consequent failure of motor movement, followed by dilatation and muscular atrophy. Distressed breathing and sudden death from heart failure may also occur, due to autonomic nerve involvement.

The common signal symptom and sequence is lameness or wing drop, followed in a few days or weeks by general paralysis, emaciation and death. In the iritis cases, the virus often seems to burn itself out, and the birds may never develop general paralysis.

Ætiology.

Much has been written concerning intestinal parasitism and catarrhal inflammation of the intestines and their relationship to fowl paralysis. Certainly these conditions have an indirect bearing on the subject, because devitalisation or abrasion of the mucous membrane occurs, and consequently the virus may obtain an easier path of ingress. Also the general resistive forces of the bird are lowered, and accordingly the invasion of the tissues by the virus is expedited. It may be stated that coccidiosis renders a flock more liable to attack by the virus of fowl paralysis, but in reality only a small percentage of birds exhibiting fowl paralysis are affected with coccidiosis or Davainea proglottina on the average. Duodenal catarrh is occasionally found in affected birds also, but it is more often brought about by faulty feeding or inherent weakness of the gastro-intestinal mechanism, and is usually coupled with stasis of bile and consequent distension of the gall-bladder.

While there is considerable evidence for the belief that biological transmission through the egg is one of the main mechanisms of transmission in fowl paralysis, it is also evident from experimental deduction, as well as from epidemiological experience, that an equally important channel of infection is through the mucous membrane of the alimentary canal and subsequent passage into the body through the lymphatic vessels, or even possibly through the nerve end plates in the mucous membrane.

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In support of this, epidemiological investigation and experimental evidence both suggest that in some circumstances fæces from birds actively affected with fowl paralysis may infect birds that are prone, and the susceptibility varies with factors such as age, strain and environmental conditions. Chickens are infected naturally during the first few weeks of life, and the more artificial the conditions of rearing the less resistant they are. This applies also to transmission through the egg, because highly artificial systems of rearing appear definitely to increase the susceptibility to invasion. Batches of chicks have frequently been found to be affected with severe fowl paralysis under four weeks of age, and have shown iritis, brain and cord involvement, and in some cases greatly thickened brachial and sciatic nerves and abdominal sympathetics, pointing pathologically to development through the egg.

In these cases particular care was taken to examine the whole course of the intestine for parasites, and in many of the cases none were found, the chicks not having been introduced to protozoal infection. Field surveys and laboratory experience combine to demonstrate that susceptibility of the chick to naturally acquired infection, which is so marked during the early weeks of life, tends to become less operative after two to three months of age, because although the maximum incidence of clinical symptoms appears usually at four to six months, the shortest incubation period is usually two months, and the virus can often remain latent or smoulder in the tissues for six months before the flame of signal signs appears, so that one can say that in the majority of cases the birds have the virus latent or incubating in their bodies at two to three months of age. Artificial infection is easier to bring about in the early weeks and months of life, but it can still be attained after six months of age, and many of the experimentally-infected birds were a year old. Natural infection, however, occurs in the young chick usually, and in the majority of cases the virus will have acquired its entrance before the eighth week, although the symptoms of disease do not make their appearance until months later.

Experimental Transmission.

Material Used.—In the experimental transmission of the disease, two hundred and forty-eight birds were used. Some of these were bought as chicks, and others at six months to a year old, from flocks which were known to be free from fowl paralysis; but the major portion of the flock was hatched from eggs obtained from clean sources. The birds were penned in small groups, or kept separately in hospital cages. Throughout the whole series of experiments one hundred and sixty normal controls were used and kept segregated in groups. Each control batch was taken from the same flock, and was of the same strain as the respective experimental group. In no instance did any of the segregated controls become affected with any symptoms of fowl paralysis.

During the early experiments birds of six to twelve months were used, and although good results were obtained with these in artificial transmission, later on birds of two to three months and even younger were utilised. The parent patho-

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logical material for transmission was obtained from thirty different birds taken from stock affected with fowl paralysis, which was sent alive by poultry farmers and killed in the laboratory. They were representative of nearly a dozen different farm flocks, and were chosen, as far as could be ascertained, for the existence of active virus. In each case selected, macroscopic and microscopic pathological verification of true fowl paralysis was made, in addition to clinical evidence of classical symptoms. As the work developed, material for re-transmission was used from artificially-produced cases, and this has proceeded in some instances as far as the third passage.

Transmission by Inoculation.—In the first four groups of six birds each that were injected, special care was taken to ensure if possible that active virus was being injected. The four batches of material for transmission were taken from four adult birds, and consisted of brain, cord and peripheral nerve in two groups, and brain, cord and spleen in the other two. Two emulsions were made in physiological saline, and two in 50 per cent. glycerol and distilled water. The material was ground in sterile mortars with quartz sand, and a 10 per cent. suspension made, which was filtered through gauze, and then slowly centrifuged for a short period. One injection of two cubic centimetres of the supernatant fluid was made intraperitoneally into each bird. Each group of six birds was injected separately from one of the four batches of pathological emulsion. The average age of the birds was just under one year.

In this first series of twenty-four birds injected, typical fowl paralysis symptoms developed in twenty cases at varying incubation periods of two months and upwards. Twelve of these twenty showed well-marked pathological signs of fowl paralysis on post-mortem examination, including enlarged peripheral nerves and changes in the spinal cord and ganglia. The remaining eight cases developed marked iritis with depigmentation of the iris and irregular constriction of the pupil, combined in some instances with varying degrees of lameness. Material from some of the twelve well-marked classical cases was re-transmitted with success. This high percentage of successful transmission has been repeated in some of the later groups, where care has been taken to use material containing active virus, and where the route of transmission has been by intra-peritoneal injection. Deduction from a varied series of cases suggests that the virus is not present at all stages of the condition in the brain and cord, but may be in the ganglia, peripheral nerves or tumour tissue if present, or *vice versa*. This, amongst other facts concerning the virus, combined with the elected transmission route, may explain the reason for some of the failures and scepticism that have attended the artificial infection efforts of some workers. At this juncture two facts may be quoted in support of the contention that the ætiological factor in the disease is a virus and not lymphoid or plasma cells associated with the infiltrative lesions.

The disease was transmitted by the inoculation of Berkefeld filtrate, even when, as will be shown, so fine a grade as the W candle was used. This, however, lessened the concentration of the virus considerably. In several instances the

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disease was transmitted by a brain and cord emulsion in a mixture of glycerol and distilled water, 50 per cent., which had been kept in the refrigerator at minus five degrees centigrade for three days. This freezing would have the effect of killing the cells, but leaving the virus intact.

Brain, cord and peripheral nerves, spleen and tumour tissue appear to be the best vehicles of virus when carefully chosen, with a view to obtaining sufficient concentration of virus. Whole blood, serum and sub-arachnoid fluid failed to transmit the disease in a small series of cases. The only consistently successful transmission route appears to be the intra-peritoneal one.

In a small group of cases inoculated intra-cerebrally, amongst failures and attendant fatalities, only one case reacted positively to a minute injection of brain and cord emulsion, and that one developed iritis. Injection by this route was not persisted in, but there appear to be possibilities. Only a small number were done in other ways, and they were all failures. These methods included subcutaneous, intramuscular, intra-neural, intravenous and intrathecal routes of injection.

Filtration.—Brain, cord and spleen emulsion was filtered through a Berkefeld W. candle, and injected intra-peritoneally into three birds. One of the three developed symptoms of fowl paralysis, including iritis. This result is striking when one considers the obstacle of the lipo-proteins of brain and cord tissue and the electro-physical difficulties of filtering a virus through such fine unglazed porcelain as the W. grade of Berkefeld candle. In another series, brain and cord emulsion from an artificially produced case was filtered through a Berkefeld W. candle and injected into three birds : one intraperitoneally, one intracerebrally, and one intrathecally, with no result. Here, again, the loss of concentration of the virus must have been considerable. Deductions from epidemiological studies and laboratory experiments led to the consideration of the possibility of the fæces being a container of virus in active cases.

Emulsions of fæces from active cases of fowl paralysis were extracted, and the extract passed through Berkefeld N. grade candles. Two c.c. of this filtrate was injected intraperitoneally into each of four young birds. Two of these developed very classical fowl paralysis in just over sixty days, and a third one in three months. These birds showed arrested general physical and ovarian development, which is often a feature of fowl paralysis in young birds. All these cases were verified pathologically, and showed well-marked enlargement of peripheral nerves and changes in the cord and ganglia. Histological sections were examined, and these showed true fowl paralysis lesions also. Taking the figures of all inoculation cases, including immunity experiments in which the virus was neutralised and many speculative attempts, two hundred birds were injected, and from these one hundred and thirty cases of artificially-produced fowl paralysis arose, a large percentage of which were verified by post-mortem examination and microscopical investigation. These figures reveal that 65 per cent., or roughly two-thirds, were positive takes.

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Incubation of the Virus.—In artificially-infected cases the shortest period of incubation for the production of marked classical symptoms was fifty-four days. The average duration of incubation was sixty-four days, but in some cases it was protracted, and the onset of definite lesions eventuated at any time up to five months. Artificial infection can be produced at any time in the first year, but more readily during the first few months of life.

Natural infection appears to take place during the first few weeks, and ceases after the first few months. Under natural conditions it would appear to be in the young chick that the virus commences to invade the nervous system, either as a result of biological transmission through the egg or by contact infection. An outstanding feature of the disease is the tendency to latency of the virus in the tissues, which accounts for the prolonged incubation periods in some cases, ranging as it does from two to six months. It would seem in some instances as though the bird was able to keep the virus locked up in certain tissue cells until some precipitating factor that lowered resistance determined the invasive distribution through the body. In some cases this latency amounted to very definite resistance, because groups of birds inoculated by the same method with the same amount and batch of virus showed varying incubation periods, while some birds appeared to remain completely immune.

Fæcal Feeding Experiments.—Various observations led to the suspected incrimination of the fæces from infected birds as a vehicle of contagion. In fowl paralysis there are a number of features which suggest infection through the intestine. Notwithstanding the scepticism that has arisen in this connection with some other virus diseases, it was decided to test the matter practically, and experiments were undertaken on this aspect. Evidence in the field pointing to the fact that if ingestion or contact infection takes place it does so in the early weeks of life, young chickens were used, taken from clean stock. These were divided into three groups of six birds each, and were fed on a good quality rearing mash containing 2 per cent. cod-liver oil, to which was added daily a small quantity of fæces from birds actively infected with fowl paralysis. These birds were isolated in cages, and in a few weeks birds in two of the groups commenced to go off their legs, but clinical examination supported the view that it was due to rickets. Consequently these two groups were removed to isolated field pens, and were given additional cod-liver oil. A number of them improved, but some rapidly grew worse, and eventually died. Three of these on post-mortem examination showed well-marked signs of fowl paralysis in the cord and peripheral nerves. The third group of six birds were Rhode Island Red chickens from a flock which has remained entirely free from fowl paralysis.

The birds comprising this group, although showing no clinical evidence of rickets, revealed at post-mortem examination a generalised softening of the bones throughout the body. They did quite well until the third month of the experimental feeding, when four of them developed clinical signs of fowl paralysis and died. On post-mortem examination definite pathological lesions of fowl paralysis

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were observed in the nervous system. The other two also died, but no evidence of fowl paralysis was seen in their case. In none of this third group were any coccidia or worms found in the intestines. In this fæcal feeding experiment seven birds out of a total of eighteen birds so fed developed pathologically verified fowl paralysis under conditions of isolation.

Contact Transmission in a Small Flock.—Fifty Rhode Island Red hens, just under nine months of age, from a flock for which it can be vouched that no fowl paralysis has occurred were ringed and placed in an extensive run on clean land. Twenty of these birds were inoculated intraperitoneally with active material from fowl paralysis cases and placed back in the run. At the end of two months the inoculated birds commenced to go down with fowl paralysis. They were left there, and a month later it was found that a number of the uninoculated birds began to show signs of fowl paralysis. This was allowed to go on for a few weeks, when it was decided to separate the affected birds. The inoculated birds that had not died from fowl paralysis or been killed for pathological purposes were removed and isolated. The uninoculated birds that showed any signs of fowl paralysis, including depigmentation of the iris or lameness, were carefully culled from those that appeared healthy. These numbered eighteen, and were put in a pen and also isolated. Six of these were very marked cases with depigmented eyes, contorted necks, and spastic paralysis of the legs, and on post-mortem examination they showed hyperæmia of the brain, softening and shrinkage of the cord in the lumbar region, enlargement of the ganglia and motor nerve trunks, and also the mesenteric nerves and abdominal sympathetic plexuses. The remainder gradually improved, including some that had a noticeable limp, and the condition became arrested. The twelve healthy birds that were left continued free from disease. The deductions from this contact experiment are various. It suggests that birds which are actively incubating the virus are capable of infecting others, in fact they appear to have as great a potentiality as birds with well-developed symptoms. Individual birds have a varying susceptibility or resistance to infection. The condition can evidence itself in transient symptoms and can be arrested. An outbreak can be stamped out if all the cases harbouring active virus are removed.

Special Aspects of the Transmission Experiments.—In the transmission experiments care was taken to exclude cases with leucotic or sarcomatous tumours, but several microscopically verified fowl paralysis tumours were used for inoculation, and proved good transmitters of the virus. An immunity experiment was carried out in one group in order to verify serologically the relationship of tumour formation in fowl paralysis to the purely neurological type of case.

A Rhode Island Red hen, aged about a year and a-half, was sent in from a laying trial with a large tumour of the left breast, which on microscopic examination was found cytologically to consist of the typical lymphoid and plasma cells that are associated with lesions in fowl paralysis. The bird had depigmentation of both eyes. There was a small tumour of the ovary, but no microscopic infil-

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tration of the nerves. The bird was bled and killed and the serum separated off. Five c.c. of this serum was injected into each of two birds, which were afterwards inoculated with emulsion from cases of fowl paralysis with nerve lesions. Five c.c. of serum from cases of fowl paralysis showing chronic nerve symptoms were injected into each of three other birds, and these birds were then inoculated intraperitoneally with ovarian and breast tumour emulsion. In all these cases no active response resulted, apparently on account of the neutralisation produced by fowl paralysis antibody. The tumour emulsion was injected intraperitoneally into one bird without the injection of serum from a fowl paralysis case. This one developed severe and typical fowl paralysis in just over two months, which was further verified by post-mortem examination. The spinal cord and peripheral nerves were involved, the latter being greatly enlarged, but there was no tumour formation in any part of the bird.

On the Aspect of Mixed Conditions.—An unusual series of artificially produced cases arose in one of the experimental groups from the intraperitoneal injection of an emulsion from a typical case of fowl paralysis of the purely neurological type without any tumour formation. The emulsion was a 10 per cent. brain, cord and spleen suspension made in the routine way, and only one injection of 2 c.c. was given intraperitoneally to each of eight birds.

Of the eight birds injected with this parent material, the first developed lameness, with no apparent nerve involvement, and a typical fibro-sarcoma of the liver in two months. The second developed fowl paralysis and a fibro-sarcoma attached to the wall of the gizzard in two and a-half months. The third developed depigmentation of the eyes in three months and a fibro-sarcoma of the heart in six and a-half months. The fourth fowl, paralysis well marked in brain, cord and nerves, and no tumour. The fifth fowl, paralysis marked in the brain and nerves, and a fibro-sarcoma of the liver. The sixth remained normal; the seventh developed fowl paralysis symptoms of wing drop and spastic gait; and the eighth enlarged sciatic nerves and tumours of the liver and spleen, which were fibro-sarcomatous. Material from these cases was re-transmitted, and with parent pathological material from the first bird a third stage of transmission was reached. From the next passage a fibro-sarcoma of the liver and pancreas arose in one case and killed the bird at the end of the second month, and in another case a tumour of the proventriculus killed the bird after three months.

Further transmission of the fibro-sarcoma of the liver and pancreas produced large fibro-sarcomata of the gizzard and mesentery, which killed the bird in five weeks, showing the exaltation of the virus. This appears somewhat difficult to explain, as there are two conditions alternating or running concurrently; but in this case the explanation probably rests in the fact that a latent virus was picked up from the injected spleen cells, which became operative, in addition to the neurotropic virus from the brain and cord tissue. The ætiological agent which produces fowl paralysis, while it can give rise to cellular proliferations assuming tumour-like form, appears quite apart from those viruses which are responsible

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for promoting the fowl sarcomata, of which we have examples in the well-known Rous chicken tumour and the Mill Hill tumours.

General Pathology.

Pathological Material Utilised.—The material studied in the investigation of the pathological lesions in fowl paralysis was obtained from the bodies of many hundreds of affected birds sent by poultry farmers and poultry organisations, together with observations on two hundred birds from the experimental flock. Naked-eye post-mortem examinations were followed by the microscopic examination of sections of diseased tissues in a large percentage of cases. Well over four hundred histological and hæmatological preparations from cases of fowl paralysis were examined microscopically.

Undoubtedly many cases of fowl paralysis will be overlooked on naked-eye post-mortem examination alone, unless the brain, cord and spinal ganglia are examined and the general features of the case are weighed up, because a considerable percentage of cases show no apparent enlargement of the peripheral or sympathetic nerves. These cases can often be elucidated by microscopic examination of the tissues. On the other hand, there are cases with a clinical history of having gone off their legs which on examination appear to have mesenteric or other abdominal sympathetic nerves which are rather abnormally large anatomically, and unless a more extensive examination is carried out a fallacious decision might be given. In such cases a heavy infestation of coccidia or worms may prove to be the factor causing simulation, or even some entirely different condition. Certainly intestinal parasites are associated with fowl paralysis in a number of cases, but there remain a large and increasing percentage where no intestinal parasites can be found. The reason for the association of the disease with intestinal parasites in the past is rational, because the main avenue of entry of the virus appears to be by the intestinal tract, and the denudation of the mucous membrane of the intestine and weakening of the general forces render natural resistance much less.

Statistically there is evidence for stating that fowl paralysis is found in a larger percentage of birds free from any intestinal parasites than formerly. Whether this is due to exaltation of the virus by passage of time or by an increased number of susceptible birds, possibly due among other factors to hyper-intensive rearing, is not certain. The incidence of infestation with protozoal and helminthic parasites is not widely divergent from that found associated with other diseases on post-mortem examination. These parasites have infested man and animals for ages past and have never yet been incriminated in producing encephalomyelitis or polyneuritis, nor from the nature of the pathological changes would it appear rational for them to do so.

Special Pathology.

Pathological Changes in the Central Nervous System.—*Lesions in the Brain.*—The substance of the brain may show hyperæmic injection or even slight hæmorrhage, and may be appreciably softened. The basal ganglia are

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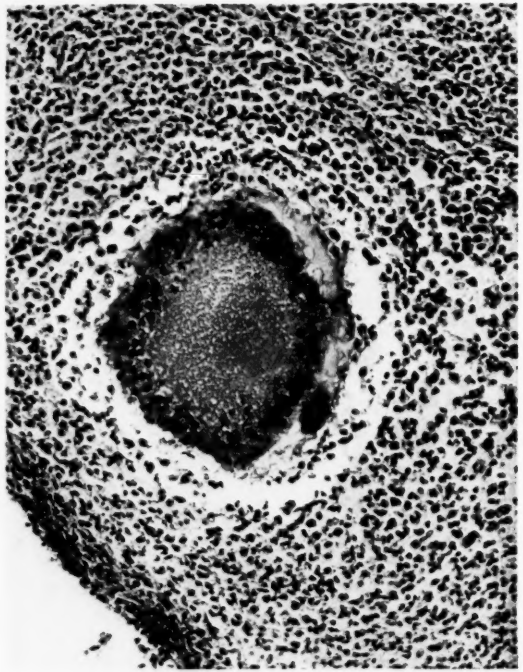

FIG. 3.—Affected ovary in fowl paralysis showing infiltration and absorption of follicle. (×310.)

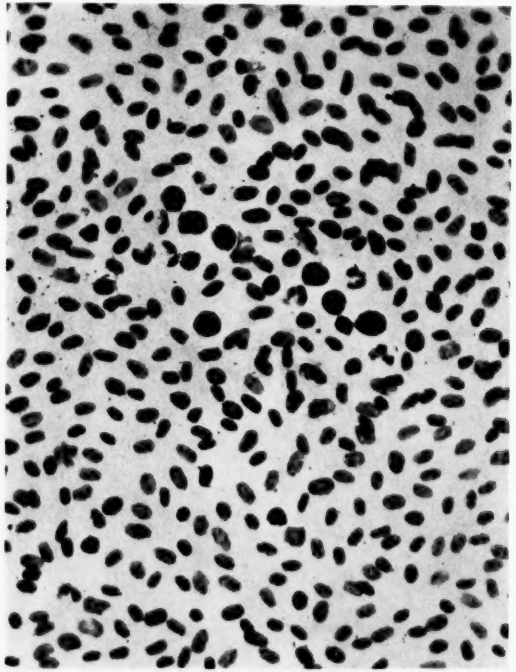

FIG. 4.—Blood cells in fowl paralysis showing lymphocytosis. (×550).

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frequently affected. Microscopically there may be slight cellular infiltration of the pia mater of the cerebral cortex. The changes in the brain are sometimes slight and difficult to find, but are mostly in the cortex, and consist of perivascular infiltration and a varying amount of interstitial infiltration with lymphoid cells.

Lesions in the Spinal Cord.—The cervical and lumbar enlargement of the spinal cord are usually the portions most affected, and particularly the latter. Varying degrees of hyperæmia with all stages of softening of the substance of the cord up to complete degeneration may be found. On microscopic examination marked changes can usually be seen in the cord consisting mainly of perivascular and interstitial infiltration by lymphoid cells. The perivascular infiltration of the sheaths of the blood vessels takes place both in the grey and white matter, and occasionally slight meningeal involvement may also be seen. The interstitial infiltration with cells occurs more constantly in the grey matter and may be diffuse or focal in character. Cytologically the infiltrating cells consist mostly of the lymphoid type with some plasma cells. Minute hæmorrhages occasionally occur. In addition to the perivascular and interstitial cellular infiltration there is degeneration of the neuron cells. The neuron cells of the ventral horns of the grey matter are sometimes affected more than those of the dorsal, but infiltrative changes are frequent in this position. One side may be involved more than another and the lesions may be confined to one level. The nerve cells show all degrees of degeneration; the nuclei may be pyknotic or eccentric, and there may be all stages of chromatolysis and vacuolation in the necrosing cells.

Lesions in the Peripheral Nervous System.—Cellular infiltration is marked in the spinal ganglia both around the nerve fibres and the nerve cells. Degeneration of the ganglion cells occurs, and perivascular infiltration of the vessels. Cellular infiltrative lesions may occur in the optic nerve with partial or total blindness, or in any of the other cranial nerves. The involvement of the eleventh or spinal accessory nerve produces the weaving and twisting of the neck which is a striking symptom in some cases. The tenth cranial or vagus nerve is very frequently affected, and when this occurs one or several of a chain of important symptoms may be produced, as this nerve is linked with the sympathetic nervous system and takes part in the control and functioning of important organs such as the lungs, heart, stomach, intestines and others. The lesions of the ganglia and plexuses of the sympathetic or autonomic nervous system are a salient and serious feature in fowl paralysis because of the conditions they produce in associated organs, such as the eye, heart, lungs, crop, stomach, gizzard, pancreas, duodenum, intestines, cloaca and others. It is extremely important to realise that many of the cases show symptoms which are not referable to the central nervous system.

Conditions such as crop binding, defective digestion, intestinal stasis, ballooning of the cloaca, respiratory distress or gasping, iritis and blindness, may be due to the lesions in the peripheral nervous system. Many of the birds which die suddenly show involvement of the autonomic nerves supplying the heart, and often

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the cellular infiltration has advanced from between the nerve filaments to invade between the muscle bundles of the organ. This involvement of the visceral organs, especially of the intestine, has undoubtedly led to the conclusion by some observers that the primary ætiology of the condition arose from intestinal lesions. The ganglia and plexuses may show great thickening to the naked eye, even though the extent of nodular formation of the pathological changes may only be evident on microscopic examination. The brachial and sciatic nerves may also be greatly thickened by massive cellular infiltration, or, on the other hand, despite wing or leg paralysis being present, they may appear normal to the naked eye, and the lesions may be found in the brain or the spinal cord. The enlargement of the nerves may be uniform, beaded or nodular to the extent of tumour formation.

The microscopic change in the nerves is of the nature of an infiltration with lymphoid or plasma cells between the nerve filaments. These cells are apparently a reactive response to axonic travel of the virus, and somewhat similar cell infiltrations are seen in the case of other neuro-diffusion viruses. Invasion by the cells becomes gradually more intense, and as a result of the massing process the axonic filaments are strangled, and cease to convey nerve impulses until ultimately they are entirely replaced by dense aggregations of cells throughout their length.

The iritis, which is such a frequent feature in the disease, is an outcome of lymphoid cellular infiltration of the sympathetic nerve innervation and the iris itself. It occurs in a large percentage of the cases that go on to general paralysis and death, but it can also appear as a more or less isolated sign in that fairly common type of fowl paralysis case in which the virus only smoulders for a while and then remains latent or dies out, leaving no other trace behind except the general spread of the condition through the flock. These cases act as disseminators of the virus, and unless the early depigmentation of the eye is noted they are a potential source of trouble.

The iris at first loses its reddish bay or similar coloured pigment round the edge of the pupil, and gradually becomes greenish or greyish. This depigmentation is due to cellular invasion, and gradually spreads centrifugally until the whole iris is involved, and the completely depigmented greyish or greenish eye is seen with a black pupil. Occasionally, owing to transient stimulation of the sympathetic, there may be dilatation of the pupil, but later there is paralysis of the sympathetic dilator fibres, so that their balancing power is lost, and the third cranial nerve constrictor fibres then overact, so that the pupil becomes narrowed to a mere slit. The infiltrating cells which invade the iris and ciliary body may ultimately cause adhesions of the iris to the lens capsule, and irregularities in the form of the narrowed pupil arise. One eye may be more affected than the other, and blindness may occur from cellular infiltration of the optic nerve itself, or partial blindness from paralysis of the ciliary body and loss of accommodation of the lens capsule.

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Changes in the Blood.

Active cases of fowl paralysis show a very definite leucocytosis, or more exactly a lymphocytosis in the blood, because differential counts in a large series of cases demonstrated that the relative increase is a lymphocytic one. This is a fairly constant feature while the disease is active, and is a useful index. The average ratio of white cells to red is about 1 to 100 normally, but in fowl paralysis this may alter to 1 to 30, the increase being due to lymphocytes. The red blood corpuscles are not altered, and the thrombocytes remain constant. There are no circulating immature red cells or immature granular white cells as seen in certain forms of the leucoses, and the bone marrow does not usually show undue erythroblastic or myeloblastic hyperactivity as is met with in those conditions.

Sub-arachnoid Fluid.—In the spinal canal the sub-arachnoid fluid becomes appreciable in amount in fowl paralysis, and is laden with lymphocytic cells, both features being abnormal.

Some Observations of the Microscopical Appearance of the Tumour-like Growths in Fowl Paralysis.

An outstanding feature in fowl paralysis is the infiltration of the nerves by lymphoid and plasma cells, but in about 10 per cent. of cases tumour-like growths composed of these cells occur, most frequently in the ovary, but also in the liver, pancreas, kidney, heart, and on occasions in almost any tissue. The typical fowl paralysis cells are of the nature of fair-sized mononuclear, basophilic, non-granular cells, rather of the undifferentiated embryonic or blast type that are usually described as lymphoid cells, and also rather larger cells that assume different shapes, but which are usually somewhat ovoid with a large cart-wheel or clock-faced nucleus designated as plasma cells. The hyperplasia commences as a cell nest, or a stasis of cells in the organ, usually perivascularly, and the invading cells press on the parenchymal cells, cutting off their nutrition, and thus causing their ultimate necrosis and absorption. The invading or infiltrating cells go on heaping up until they produce a tumour-like growth replacing the tissues of the invaded organ. These cellular hyperplasias in the organs sometimes lead to confusion with one or other of the leucoses, and the mistake can take place more readily in some cases owing to the fact that in the later stages of this condition the bird may go off its legs through weakness.

The Genetic Aspect of Fowl Paralysis.

There is an accumulation of evidence which points to transmission through the egg in some circumstances, but for this to take place the virus must be present in the tissues, whether it be latent, smouldering, or acutely active in the transmitting cockerel or hen. A particularly responsible aspect is demonstrated in the case of cockerels, which being sometimes more resistant, may suffer from a mild infection and be capable of transmitting the active virus. Epidemiological investigation has shown repeatedly that on farms previously free from fowl paralysis the chicks hatched from bought eggs have developed fowl paralysis, and

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on further inquiry it has been found that there was active fowl paralysis in the parent flock from which the eggs came.

Transmission Through the Egg.

A genetical experiment was undertaken to obtain data on this aspect. Batches of one hundred eggs were obtained, each from different sources: (1) From a flock in which fowl paralysis had been present the previous year, but had died out. (2) From a flock in which there was a considerable amount of active fowl paralysis. (3) From a flock which was free from fowl paralysis, and which was even considered resistant to the condition. Rearing conditions were identical. An analysis of the findings after six months is as follows: In the first group of chickens there had been no development of fowl paralysis. In the second group there were 25 per cent. of deaths attributed to histologically verified fowl paralysis. In the third group there was no incidence of fowl paralysis. This in itself rather supports the belief of biological transmission through the egg. In the field study of flocks there is a good deal of evidence to support this view. Turning to the pathological consideration, it will be noted that of the lesions found outside the nervous system in fowl paralysis, cellular infiltration of the ovary is the commonest, so that there is also pathological ground for suspecting egg transmission. Certain environmental conditions undoubtedly increase the susceptibility of chicks and determine the invasion or the advance of the virus in the tissues, as well as assist in the spread of the condition amongst the young birds. Overcrowding in an atmosphere deficient in oxygen with the tropical conditions of high temperature and humidity, such as maintains in some intensive rearing systems, undoubtedly increases the susceptibility to virus advance by lowering the resistance of the tissues. Evidence for the belief that strains exist which are more resistant to fowl paralysis than others has been tendered by Biely, Palmer and Asmundson (1932). Statistics support the contention that birds which are brought up as close to the postulates of nature as possible, and are early on range, are more resistant than the artificially-forced and pampered flocks. More work to elucidate genetical problems in fowl paralysis remains to be done, and is being undertaken in the near future in these laboratories.

Epidemiological Investigation of the Causative Factors.

From a survey of a considerable number of flocks there appeared to be almost invariably a fairly constant sequence in the chain of circumstances under which fowl paralysis would appear on a farm. The alternative factors in the production of an outbreak were, in order of frequency: (1) The importation of cockerels from a flock where fowl paralysis existed either in its open paralytic form or in one of the less apparent forms of sympathetic nerve or eye affection. (2) The buying of eggs from infected sources. (3) The buying of day-old chicks hatched from infected eggs. (4) The intensive rearing in battery brooders of infected and non-infected chickens together. (5) The mixing of imported affected chickens on range with chickens originally free from the disease, with less incidence than in the case of those reared in battery brooders.

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It will be seen that the buying of eggs or chickens is beset with considerable danger if they come from infected sources, because although the chickens appear perfectly well, the virus may be latent or incubating in their tissues, and paralysis may make itself apparent at any time up to the age of six months. The question of the viability of the virus in the bird's tissues is a difficult one, and can be better determined in any case by the success or non-success of transmission rather than by clinical examination. Efforts to devise a serological test of the nature of a sero-flocculation with a variety of chemical reagents and biochemical antigens acting on serum from cases of fowl paralysis has so far not given completely consistent results. Well-developed cases can usually be judged from the clinical signs, and it is problematical yet whether any sero diagnosis test can be evolved which would give a reliable indication as to the existence or absence of virus in any bird at any particular stage. Further work on this subject is, however, proceeding at the present time.

Infection of Land by Virus.—The question of infective contamination of the houses and ground by the fæces from infected birds is one of paramount importance, because there is epidemiological and experimental evidence to suspect the fæces as a vehicle for the virus. How long the land remains infected will depend partly on the mass amount of infection and the type of soil, but above all on the physico-chemical properties of the virus in question. Like some other viruses, it appears to resist desiccation, freezing and storage in glycerine. It is killed by antiseptics, but the difficulty is that they have to be adequately concentrated and the contact sufficiently prolonged to attain the viricidal action. The application of dry slaked lime to the land is not a very efficacious measure. It is much better to use a solution of quicklime in water, but it has a deleterious effect on vegetation. This can also be used for swabbing out houses and floors, or for this purpose solutions of formalin (1/100) or crude cresols (1/100) may be substituted. Painting with creosote is a useful measure. Despite the resistive features of most viruses to drying and other physical processes, it seems reasonable to assume that the virus would have died out on infected land which had been treated and left fallow within a few months. The ultra-violet rays of the sun have a determined viricidal power.

Prophylaxis and Control of the Condition.

In the case of a neurotropic virus such as this one, which causes degeneration of the nerve cells as well as producing cellular proliferation as a reaction by the tissues to the diffusing virus, there is little chance of successfully treating and stemming the advancing toxic process unless the resistance of the individual animal is normally peculiarly high, or is artificially stimulated. Rationally it cannot be expected to get the same dramatic relief from symptoms that occur in rachitic weakness or in polyneuritis due to avitaminosis by merely feeding the requisite vitamin. Experience of parallel conditions in the central nervous system of man and mammals renders it unlikely that any specific chemotherapeutic substance known at present can be used to penetrate to the central nervous

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system and save the nerve cells and axonic filaments. Once the virus has entered the tissue cells, and particularly when it has started its attack on the nerve cells, the invasion is difficult to check even by the use of hyper-immune serum. The transient passive immunity which it endows may be useful for in-contact cases, but it is impracticable, particularly in the case of large flocks. The more rational method is to make birds proof to infection by active immunisation, commencing preferably at an early age. This was achieved over six months ago in a series of cases by means of serum-virus consisting in the simultaneous injection of properly balanced amounts of immune serum and live virus. However, there are economic difficulties in the application of serum-virus, and consequently work was directed on the technical perfection of a " vaccine-virus," which consists in the injection of an inactivated antigenic virus emulsion followed by a modified live virus. The most important factor is that the antigenic power of the vaccine must be sufficiently potent to provoke a satisfactory immune-body response in the first place.

Virus inactivated chemically by varying strengths of formol, formol-glycerol, phenol, phenol-glycerol, chloroform, ether and other reagents, or inactivated physically by various forms of irradiation or other physical factor, are difficult to standardise in antigenic value, and are unstable as regards conservation of these properties. Unless their virus-content is sufficiently potent, antibody production is only procured after repeated doses, or even the injection of desiccated live virus may be required to produce a solid immunity. Extensive research is being carried out at the present time on this subject, and will form the basis of a further communication in the near future.

Vaccine-virus immunisation would be better applied to chicks as young as possible in order to prevent or stamp out the invasion and incubation of virus, but it could be of protective value at any age. Naturally, the first thing to do on a farm harbouring fowl paralysis is to cull all infected stock, remembering the eye cases and sympathetic nerve cases. Then disinfection of the houses and land has to be undertaken either as a whole or in sections. Efforts should be made to create a foundation breeding stock free from taint of infection, and if possible of a resistant strain, and to this end the invulnerability of this foundation breeding stock might be rendered more certain by immunisation, and likewise the certainty of freedom from the condition of their progeny might be promoted by a similar precaution.

On surveying the situation it is a very noticeable fact that a large percentage of the flocks in this country are heavily infected with fowl paralysis, and until scientific measures are taken advantage of to check the onset of this disease there is no doubt that it will blaze or smoulder through the young flocks, according to their degree of susceptibility, each rearing season with compound interest.

Summary and Conclusions.

(1) Experimental evidence proves that the ætiological agent in this disease is a virus and not a cell particle. This is clearly demonstrated by its ability to pass through a Berkefeld filter and to withstand storage in glycerol at minus five degrees

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centigrate for several days. (2) The virus in fowl paralysis acts primarily on the brain, spinal cord and peripheral nervous system. (3) The main symptoms may not be referable to a central origin, but may be produced by lesions in the autonomic nervous system associated with various organs. (4) The virus may provoke a cellular infiltrative hyperplasia of tumour-like proportions in various visceral organs, particularly the ovary. (5) Lymphoid cellular lesions of the iris are frequent. (6) Lymphocytosis in the blood is usually present in active cases. (7) The virus can apparently remain latent in the tissues for many months until factors determine its advance, or it can smoulder for some time, producing merely incipient or transient symptoms which may escape detection and yet act as a focus of infection, transmitting the condition to the progeny through the egg or to young normal birds by contact. On the other hand, it may progress very quickly, with few signs on post-mortem examination except those in the brain and spinal cord or the autonomic ganglia. (8) The average incubation period is usually over two months. (9) Natural infection appears to take place through the egg or by ingestion, the path of ingress presumably being through the mucous membrane of the alimentary canal. The fæcal excretions would appear to convey infection during the incubation and some aspects of manifestation of the virus. (10) Natural infection occurs during the first few months of life, and susceptibility decreases after six months of age. (11) The maximum incidence of onset of symptoms occurs usually at three to six months of age in natural infections. (12) Artificial infections can be produced at any time up to a year of age. (13) Various factors, such as environmental conditions in rearing and infestation with parasites amongst others, help to precipitate the invasion of the tissues by the virus. (14) The importation of breeding stock into a flock, particularly infected cockerels, often determines the ultimate onset of an outbreak amongst the progeny. (15) Apart from care in making sure that the foundation breeding stock is clear from the disease and that chance of contact infection is eliminated, measures taken to actively immunise the progeny give promise of ultimately providing a method of control.

APPENDIX I.

Incidence of Fowl Paralysis in Breeders' Stocks as shown by Deaths at Laying Trials and Verified by Microscopic Examination.

Owner A.—Six out of ten deaths due to fowl paralysis. *Owner B.*—Four out of five deaths due to fowl paralysis. *Owner C.*—Four out of seven deaths due to fowl paralysis. *Owner D.*—Five out of eleven deaths due to fowl paralysis. *Owner E.*—Eight out of eight deaths due to fowl paralysis. *Owner F.*—Six out of eight deaths due to fowl paralysis. *Owner G.*—Four out of four deaths due to fowl paralysis. *Owner H.*—Three out of four deaths due to fowl paralysis. *Owner I.*—Two out of three deaths due to fowl paralysis. *Owner J.*—Two out of two deaths due to fowl paralysis.

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APPENDIX II.

THREE CASES OF TRANSFER FROM ADULT TO PROGENY, BY THE IMPORTATION OF COCKERELS FROM AN INFECTED STOCK, PRODUCING AN OUTBREAK OF FOWL PARALYSIS ON A FARM HITHERTO CLEAR OF THE DISEASE.

Case A.—Breeding stock for season 1932–33: Sixty per cent. 1931 hens (R.I.R.); 40 per cent. 1932 pullets, daughters of 1931 hens above.

Mating 1.—To R.I.R. cockerels purchased from Breeder X as eggs, in January, 1932. Mortality amongst cockerels, 25 per cent.; but only one post-mortem examination was made, and this revealed fowl paralysis. Breeder X stated that there was no disease on his farm, but subsequently it transpired that he lost 2,000 chicks from various causes.

Hatching.—Fertility below normal, hatching being slightly above. Chicks were weak and below standard.

Rearing.—Extensive from brooder house. Losses were 15 per cent. Food given was a proprietary brand of repute.

Advanced Rearing.—Food from same source. At three months of age chicks showed signs of paralysis. Both sexes equally affected. More evident when the pullets reached laying age. Total losses during seven months, 20 per cent.

Mating 2.—In February the same hens were mated to White Leghorn cockerels. No fowl paralysis has appeared. In breeding pens where cockerels were used other than those hatched from the eggs supplied by Breeder X, no fowl paralysis appeared. Fowl paralysis was never seen on this farm prior to this breeding season, 1932–33.

Case B.—This farm has been established for five years, and has previously been free from infection. For the breeding season 1932–33 cockerels were bought as eggs from Breeder Y. The result has been widespread development of fowl paralysis amongst the progeny from the ages of three to six months. Several of the patent cockerels showed signs of fowl paralysis, and the diagnosis was confirmed on post-mortem examination.

Case C.—The farm was established in 1928, and no trouble was met with until the hatching season 1932–33. Cockerels were bought for the 1932–33 season from Breeders A, B and C. Breeder A's R.I.R. flock is known positively to be infected with fowl paralysis, while Breeders B and C are free from the trouble.

Breeding.—Fifty per cent. pullets and 50 per cent. hens. Pullets are mated with cocks, and hens with cockerels. Progeny of pullets bred from as pullets, which are early hatched and moulted out before being put in the breeding pens.

Hatching.—Is by small hot-air machines.

Rearing.—Chicks go to hovers in brooder house, 500 size, accommodating 300 to 400 chicks. They are let out on the grass as early as possible, usually at

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the sixth day. The first symptoms appeared at eight to ten weeks of age, and birds four to five months of age were still developing fowl paralysis when last observed. It was noted that the chicks hatched from pens headed by cockerels bought from Breeder A have developed fowl paralysis to a greater extent than is the case with the others.

APPENDIX III.

Contact Transmission from Affected to Normal Stock.

Farm previously free from trouble. Season 1932–33 : day-old chicks were bought from large battery brooder plants, and later the chicks were mixed with chicks hatched on the farm. Battery brooder chicks went down with fowl paralysis, and later to a lesser extent fowl paralysis developed amongst the home produced stock.

APPENDIX IV.

Onset of Fowl Paralysis in Clean Strain placed on land where an outbreak had occurred.

This farm experienced a severe outbreak of fowl paralysis, and the owner spread the fæces from the birds over the land with the idea of fertilising it. Then, with a view to stamping out the epidemic, he culled a great part of the stock and replaced it by day-old and month-old chicks bought from clean stock. These he kept apart from the adult stock, but they ranged on land that had recently been spread with the fæces of the affected birds. At three to four months of age they began to exhibit signs of fowl paralysis, and the outbreak re-commenced.

Acknowledgments.—Thanks are due to the Director of the Laboratories for the unsparing material efforts he has made to promote and engender the work of the investigation in every way, and further for the use of epidemiological data compiled from a wide experience. Additional thanks are tendered to Mr. N. D. Constantine, Research Assistant, for his efficient co-operation in the work, and for the excellent pathological preparations he has produced.

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THE QUANTITIES OF VITAMIN A REQUIRED BY PULLETS FOR MAINTENANCE AND EGG PRODUCTION.*

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SOME work has been reported [4,2,1] that shows the effect of varying quantities of vitamin A in the feed on the health of laying hens, on their body weight, on the number and size of the eggs laid, on the fertility, hatchability and vitamin A potency of the eggs, and on the viability of the chicks hatched. No previous work has been found that gives the quantity of vitamin A required for production of eggs or maintenance of laying hens. The work conducted at the Texas (U.S.A.) Agricultural Experiment Station, and reported in this paper, shows the effect of feeding measured quantities of vitamin A to pullets upon the health of the birds, the number of eggs laid, and the vitamin A content of the eggs ; it also indicates the quantity of vitamin A necessary for maintenance of the pullets and for the production of eggs rich in vitamin A.

White Leghorn pullets that had been raised on a standard ration, including free access to green feed, were divided into three groups of thirty-six pullets each. The pullets were approximately six and a-half months of age when started on the experiment, which lasted six and a-half months. Twenty-two days after the experiment started one pullet of poor quality was removed from each group. The group called in this paper the yellow-corn group was fed a mash containing 20 per cent. each of ground yellow corn, wheat gray shorts, wheat bran, ground oats, and meat and bone scrap. Their scratch grain consisted of whole yellow corn. The mash fed the group called the mixed-corn group was the same as the one just mentioned except that one half of the ground yellow corn was replaced by an equal amount of ground white corn. The scratch for this group was composed of equal parts of yellow corn and white corn. The group called the white-corn group received white corn entirely instead of yellow corn ; otherwise the ration was the same as the others. No cod-liver oil, alfalfa meal or green feed was fed any of the pullets. All groups were confined on cement floors.

The amount of vitamin A in samples of the yellow corn fed these pullets, and in the yolks of representative lots of eggs produced by the pullets of the different groups, was determined by the Sherman-Munsell unit method, using the rat as the test animal.[3] A rat unit is an average gain of 3 grams a week for eight weeks. Vitamin A was not determined in the ingredients of the feed other than yellow corn. The other ingredients of the mash contained almost negligible amounts of vitamin A, and the three rations were identical except for the corn ; however, the vitamin A in these other feeds was not disregarded in the calculations, and does not invalidate the conclusions drawn from this study. The average

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amounts of vitamin A consumed daily in the yellow corn was approximately 270 units for the yellow-corn group, 120 units for the mixed-corn group, and none for the white-corn group.

The amount of vitamin A in the rations had a direct effect on the mortality of the birds. The mortality was very high in the white-corn group: one pullet died the thirty-third day of the experiment, two others died before the end of the second month, three died in the third month, seven died in the fourth month, twelve died in the fifth month, four died in the sixth month, and two died the last half-month of the period. Thirty-one pullets died out of thirty-five during the entire period. In the mixed-corn group two pullets died the fourth month, and two died the last half-month of the experiment. In the yellow-corn group one pullet died the third month, another in the fourth month, and a third during the six month. The body weight of the pullets was also affected by the rations. At the close of the experiment the yellow-corn group averaged 25 per cent. more in weight than the white-corn group. The mixed-corn group weighed nearly 17 per cent. more than the white-corn group.

The rations had a direct effect on the number of eggs laid. This became evident by the end of the second month of the experiment. During the last four months of the experiment the yellow-corn group laid approximately 65 per cent. more eggs than did the white-corn group, and the mixed-corn group laid 35 per cent. more eggs than did the white-corn group.

The vitamin A content of the eggs showed definitely as the experiment advanced that none of the rations contained enough vitamin A for high egg-production. The vitamin A content of the eggs from all of these groups became lower as the experiment progressed. The eggs from the yellow-corn group held their vitamin A content at 20 units per gram of yolk for about two months, and then gradually dropped to 5 units of vitamin A per gram by the close of the experiment. The vitamin A content of the eggs from the other groups had started to drop before the birds had been on experiment seven weeks. The content gradually dropped until it was approximately 5 units per gram of yolk before the close of the experiment.

Calculations were made on the quantity of vitamin A required to supply the vitamin A in the eggs, by a study of the vitamin A intake of the yellow-corn and mixed-corn groups and the vitamin A found in the eggs. The average amount of vitamin A in the yellow corn fed each pullet in the yellow-corn group was 53,394 units during the experiment. There were 23,991 units of vitamin A per pullet in the yellow corn fed the mixed-corn group. The difference between the vitamin A fed in the yellow corn to these two groups was 29,403 units per pullet. There were 14,018 units of vitamin A per pullet in the eggs from the yellow-corn group as compared with 9,342 in the mixed-corn group. The difference in vitamin A between these groups was 4,676 units. Thus, 29,403 units of vitamin A fed to the yellow-corn group in excess of the amount fed to the mixed-corn

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group produced 4,676 more units of vitamin A in the eggs than were in the eggs of the mixed-corn group. This is a utilisation of 15.9 per cent. of the vitamin A for the eggs. This means that one unit of vitamin A in the egg requires 6.3 units in the feed in excess of maintenance requirements. This method of calculation allows for both the vitamin A used for maintenance and that stored in the bodies of the pullets at the beginning of the experiment, as well as for the small amounts in the feed other than the yellow corn. Sufficient allowance may not have been made for maintenance requirements, but the figures used are the closest that can at present be secured for the utilisation of vitamin A for storing in the egg when fed in fair amounts, but amounts still insufficient for maximum storage.

The estimation of the amount of vitamin A required to meet the maintenance requirements during egg production is not as certain as for the vitamin A in the eggs, on account of the wider variation in the amount of vitamin A stored in the bodies of different pullets, as shown by the survival periods of the pullets in the white-corn group. The average vitamin A content of the eggs laid by the pullets in the mixed-corn group for the entire experiment was 9,342 units, of which it is assumed that 8,843 units may have come from the body of the pullet, since the birds of the white-corn group produced this amount in their eggs without receiving yellow corn. The difference (499 units) between the amount of vitamin A in the eggs of the two groups required 3,144 units (499 units $\times$ 6.3) of vitamin A in the feed. When these 3,144 units of vitamin A are deducted from the 23,991 units fed the mixed-corn group during the period, there remain 20,847 units for maintenance for 199 days, which is approximately 105 units of vitamin A per pullet per day. The data of this study show that it requires approximately 1,365 units of vitamin A per day for a laying pullet for maintenance and production of eggs at the rate of 20 eggs per month. These figures are based on egg yolk weighing 15 grams each, and containing 20 vitamin A units per gram. These 20 eggs would contain 6,000 units of vitamin A, and would require 37,800 units (6,000 units $\times$ 6.3) of vitamin A in the feed, or 1,260 units per day. This amount, plus the 105 units per day for maintenance, would amount to 1,365 units per day for maintenance and for an average production of 20 eggs per month.

These estimates of the vitamin A requirements for laying pullets are based upon one experiment, and, of course, require further confirmation. They are, however, in line with the work of Bethke, Kennard, and Sassaman,[1] who state that hens fed a basal diet containing 30 per cent. of yellow corn supplemented with alfalfa meal, fed *ad libitum*, produced eggs containing only one-fifth as much vitamin A as when receiving cod-liver oil or having access to blue-grass pasture. It has been pointed out that laying hens require large amounts of vitamin A for maintenance and the production of eggs high in vitamin A. Feeds excellent for maintenance of growth may not supply the large amounts of vitamin A required for the production of such eggs. The quantities of yellow corn, ordinary alfalfa

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meal or alfalfa leaf meal, or other sun-cured green feeds eaten by laying hens, do not supply sufficient amounts of vitamin A. Green grass or green growing plants seem to be best suited to supply the large amounts of vitamin A needed.

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UNEXPECTED RESULTS OF MATINGS INVOLVING SEX-LINKED CHARACTERS.*

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RECENTLY I have had the opportunity of examining three instances in which unexpected results were obtained by breeders who were applying the facts of sex-linked inheritance to commercial poultry breeding. This practice is widespread, and every year witnesses the hatching of thousands of chicks the plumage character of whose mothers was genetically silver, whilst that of their sires was genetically gold. This mating is relied upon to yield, in more or less equal numbers, gold daughters and silver sons, and since these colours can be recognised and distinguished in the newly-hatched chick, it is possible to identify the sex of the individual by reference to the colour of its down.

Dominant silver and its alternative recessive gold form a pair of sex-linked characters, that is to say, the hereditary factors or genes corresponding to them are resident in the X-chromosome of which the male has two, the female but one. Since in any one X-chromosome there can be either the gene for silver or else the gene for gold, the female possessing only one X-chromosome must necessarily be genetically pure-breeding for either silver or for gold, whereas the male, having two X-chromosomes, can be a pure-breeding gold, with two genes for this character, a pure-breeding silver, with a gene for silver on each of his X-chromosomes, or else a silver (this being dominant) carrying gold.

The female chick receives her single X-chromosome by way of the sperm from her father. Associated with this paternal X-chromosome there is a Y-chromosome derived from the mother and carrying no genes for the sex-linked characters. A daughter, therefore, receiving her X-chromosome from her father, will neces-

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sarily exhibit those sex-linked characters which he exhibited. The male chick receives one X-chromosome from each of his parents, and therefore will exhibit the dominant member of each pair of sex-linked characters exhibited by the parents. The distribution of sex-linked characters is determined by the distribution of the X-chromosomes. The regularity in the distribution of the former is determined by the precision of the mechanism which is distributing the sex-chromosomes. Manifestly, unless the new individual derives its X-chromosomes or chromosomes from the usual source, the sex-linked characters that it exhibits may be exceptional and the bird will not obey the rules of sex-linked inheritance. Furthermore, the plumage colour characters of the parents must be genetically silver and gold and the transmission of these must be uncomplicated by the presence of non-sex-linked factors which inhibit the expression of gold or which yield colours such as chestnut or bay, which look like gold but do not behave like it in inheritance.

(A) **Faults in the Sex-Chromosome Distributing Mechanism.**

In the case of the usual laboratory experimental material of the geneticist it has been noted that among the offspring of a mating involving sex-linked characters not less than about 1 in 2,000 individuals may be expected to exhibit the outward and visible signs of a faulty distribution of the sex-chromosomes. The commonest fault is what is known as non-disjunction. In the cock there are to be found in the nucleus of every cell of the body and of the unripe sperm two X-chromosomes. But during the final stages of ripening or maturation of the sperm these two X-chromosomes disjoin or separate, and the result of this is that in every ripe functional sperm there is but one X. In the hen there are to be found in the nuclei of the body cells and in that of the immature egg an X together with a Y-chromosome. As the egg ripens the X and the Y disjoin, so that the functional egg, ready for fetilisation, has either an X or else a Y, but not both. But consider what would happen if in a ripening sperm the two X-chromosomes failed to disjoin, so that the finished sperm came to possess two X's instead of one. If such a sperm should meet and fertilise an egg containing a Y-chromosome (note that this Y carries no genes corresponding to the sex-linked characters) then an XXY individual would result, and the sex-linked characters exhibited by this cock (a male because there are two X-chromosomes) would be those of the father. Thus, a gold cock mated to silver hens could produce a gold son, *i.e.*, one who disobeys the rules of sex-linked inheritance. Furthermore, this gold son mated to silver hens would, in his turn, produce, in addition to silver sons and gold daughters, exceptional gold sons and silver daughters. The reasons for this are given in the following scheme :

S = the gene for dominant sex-linked silver ;
s = the gene for recessive sex-linked gold ;
X = the X-chromosome : Y=the Y-chromosome ;
( ) = the included gene is resident on the included X.
A non-disjunctional gold cock × a silver hen
(sX) (sX) Y (SX) Y

The normal gold cock (sX) (sX) would produce sperm all of a kind (sX), but this cock produces four kinds in respect of the contained sex-chromosomes. The two X's can disjoin, and with one of them the Y will go, or else the two X's will remain together and the Y will pass into an egg alone. It is established that the two X's disjoin more commonly than not.

The hen will yield two kinds of eggs:

eggs	(SX)		(sX)	
	Y		(sX) Y	sperm
			(sX) (sX)	
			Y	

Fertilisation of these two kinds of eggs by these four kinds of sperm will yield the following kinds of individuals:

(1)	(SX) (sX) ...	a silver	male	expected
(2)	(SX) (sX) Y ...	,,	,,	,,
(3)	(SX) (sX) (sX) ...	,,	,,	,,
(4)	(SX) Y	,,	female	exceptional
(5)	(sX) Y	gold	,,	expected
(6)	(sX) YY ...	,,	,,	,,
(7)	(sX) (sX) Y ...	,,	male	exceptional
(8)	YY	does not appear.		

No. 4, the exceptional silver female, is exceptional because she receives her Y-chromosome from her father (which is impossible in the case of the normal male) and her X-chromosome from her mother (instead of from her father). No. 7, the exceptional gold male, is exceptional because he receives both his X-chromosomes from his gold father instead of one from his father and the other from his mother who was a dominant sex-linked silver. Such a bird, a gold cock out of the mating sex-linked silver hen and sex-linked gold cock, has been examined during the last year, and a complete account of his breeding record and of the sex-chromosome constitution of his offspring is to be found in the *Proceedings of the Royal Society of Edinburgh*, XLIII, Pt. II, No. 8, 1933.

(B) The Expression of Sex-linked Silver and Gold Complicated by the Simultaneous Expression of Sex-linked Barring and Non-barring.

A well-known breeder in the North of Scotland who had used the White Wyandotte Rhode Island Red mating for several years to obtain gold daughters and silver sons, found during the last two years that in addition to these typical golds and silvers a number of black chicks appeared. She was able to classify them into two distinct groups, since some of them had a russet tinge on the face and wings whilst the rest were silvery-faced and had on the top of the head a distinct white spot of varying size. Naturally enough, I assume that these black chicks were unbarred gold females and barred silver males. I obtained some two dozen of these chicks from her and reared them. The silvery-faced chicks with the occipital spot are now barred silvers, and the russet-faced ones are non-barred

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golds with a pheasant-like plumage. In this stock two pairs of sex-linked characters were present, silver and gold, barred and non-barred, and thus the presence of these black chicks among the more distinct golds and silvers was explained.

(c) **A Silver which is an Inhibited Gold.**

Clear-cut results will not be obtained if the hens used are not pure uncomplicated sex-linked silvers. There is an autosomal gene which inhibits the expression of sex-linked gold, and which produces in the presence of this gold a range of colours varying from reddish yellow to silver. Manifestly, if the silvers used are of this kind, the results will seem to deny the principles of sex-linked inheritance. This autosomal modifier of gold is discussed in a paper by L. W. Taylor in the *Proceedings of the International Congress of Genetics*, 1932.

THE VENOUS SYSTEM OF THE FORE-LIMB OF THE DOMESTIC FOWL IN RELATION TO BLOOD TESTING.

By W. P. BLOUNT, F.R.C.V.S.

Poultry Health Laboratories, Moulsford, Wallingford, Berks.

It is well known that the application of the blood agglutination test for the eradication of Pullorum disease in poultry has reduced chick mortality considerably. The procedure is simple, but unfortunately the assistance of veterinary surgeons is not invoked by the poultry keeper as frequently as desired, and moreover, from letters received from practitioners, it is evident that a number do not comprehend fully the method. Boxes in units of 50 or 100 tubes for the collection of blood may be obtained free from the majority of Poultry or Veterinary laboratories. Each blood-testing outfit contains, in addition to its tubes, a needle for the puncturing of the vein, list of instructions, and "flock sheet." This latter is used to indicate which birds have been bled, into which tube each sample of blood has been put, and also to record certain particulars concerning the "leg band" number, breed and pen to which each bird belongs.

It is customary to obtain the requisite quantity of blood (about 2 c.c.) from one of the wing veins, and as a rule the vein which passes over the medial aspect of the elbow-joint is chosen. Unfortunately, the flow of blood obtained by puncturing the vein at this site is frequently unsatisfactory, since the rate is often very irregular and the necessary quantity of blood difficult to obtain unless both wings are utilised. In order to overcome this difficulty the writer has carried out certain dissections of the parts concerned, and it is now possible to indicate clearly a better site for venesection. In actual field practice it is possible for an operator

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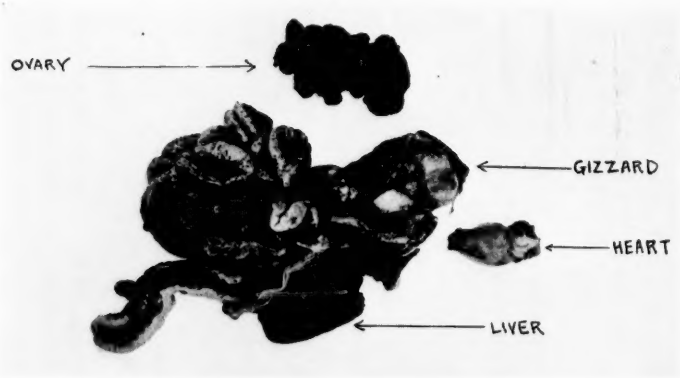
Photograph illustrating the superficial veins on the underside of a domestic hen with reference to blood testing. Note the old site for venesection where the vein crosses the elbow joint, and also the new site which is lower down the arm at the junction of three veins, two of which can be seen.

(Article by W. P. Blount, F.R.C.V.S., *page* 480*)*



Ovary, heart and intestinal organs of fowl affected with melanosis.

(Reproduced by kind permission from the Report of The Poultry Pathological Research Laboratory.

A case of fowl paralysis.

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to bleed, with the help of two assistants (one to catch and the other to hold the birds), at least seventy-five birds an hour. It will be seen from the diagram that the usual place for puncturing the vein is immediately anterior to the elbow-joint, *i.e.*, at a point where the vessel (basilic vein) passes to lie between the tendons of origin of the flexor carpi ulnaris and pronator brevis muscles.

The basilic vein may be seen passing along the ulnar border of the forearm, where it receives branches from the wing feathers. Turning ventrally, the vein crosses the elbow-joint to run anterior and parallel to the distal half of the humerus. About midway down this bone the vein commences to run caudally, finally to disappear at the back of the shoulder joint, where it soon passes to join the axillary vein. Throughout this course the vein remains superficial and subcutaneous in position, hence its relatively prominent situation and attraction for the poultry keeper. In examining the living animal another blood vessel running more or less parallel (though ventral) to the ulnar may be determined. At first glance this vessel (*V. ulnaris profunda*) appears to terminate by emptying its contents direct into the basilic vein, but in point of fact it merely anastomoses with the *V. basilica*, and then disappears in conjunction with the ulnar artery under the medial (humeral) aspect of the *M. biceps brachii*. At this stage it is well to note the following area bounded posteriorly by the humerus, anteriorly by the tendon of insertion of the *M. biceps brachii*, and dorsally by the tendon of origin of the *M. pronator brevis*. This triangle encloses an important group of blood vessels, and it is in this region that it has been found best to puncture a vein prior to bleeding. Here the basilic vein receives not only the anastomosing branch from the ulnar vein, but also an even larger vessel—the deep radial vein. At this point, therefore, a large composite vein commences, which if punctured will bleed freely and easily. It is commonplace to apply gentle pressure at the base of the arm of the bird with the thumb of the left hand, but it is clear that to be really effective in acting as a tourniquet the pressure should be applied towards the back of the shoulder joint (*see* diagram).

Not infrequently poultry keepers have experienced trouble with "blood blisters," due to the fact that subcutaneous hæmorrhages have occurred immediately following the puncture of a vein. This automatically stops the flow of blood, and if using the old site for venesection renders the limb useless. If the new method be adopted blood blisters will be found to be rare, because the flow of blood is strong and rapid. Further, on those few occasions when such faults do occur it will be appreciated that it is still possible to resort to a site more distal from the body, but using the same wing vein.

It is the work of the practitioner to ascertain from his client the number of birds to be bled, then to obtain the necessary tubes, etc., from a laboratory, and finally to bleed the birds and send the blood samples to the laboratory, where the necessary tests will be carried out for a fee of 2*d*. per sample. Veterinary surgeons usually charge their clients $\frac{3}{4}d$. for each bird bled, plus travelling expenses.

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BLACKHEAD IN TURKEYS.

By G. MAYALL, M.R.C.V.S.
Bolton, Lancashire.

THE man who can produce a plan for the prevention and cure of this disease in turkeys will deserve a seat in Paradise in the mind of poultry keepers. It is a fell disease that causes the deaths of hosts of young turkeys in a season. Most recent research attributes the malady to a flagellate protozoon parasite (hence *Flagellosis* of turkeys), the eggs of which are taken up by the turkey, and being swallowed, are incorporated in the common pin-worm—*Heterakis gallinæ*—of the cæca. The parasites develop in the intestinal lumen, especially in the blind gut, and through the small lesions caused by the worm enter the tissue of the muscular coat of the bowel and are conveyed by the blood stream to the liver, or may perhaps penetrate directly from the intestinal lumen through the gall duct to the liver. It would appear, therefore, in any attempt to prevent infection, turkeys should be kept free from worms, and the haunts of protozoon parasite attacked. The parasite is favoured in its development by moisture and warmth, and so ponds and puddles should be absent on turkey grounds. Hens may be carriers, and should never run with turkeys. Starlings, rats and mice should be got rid of. Milk feeding in early days fortifies the system against attacks of the parasites. Young turkeys during the first weeks should not run free, but be kept penned (Heelsbergen). In America young turkeys are reared on a rotation system, and every week they are driven on to a fresh plot of land, and the vacated land may be left vacant for six months. (This supports the axiom that turkey breeders, to be successful, must have extensive range ; other advantages are ample shade and a dry subsoil.)

The disease, as stated, being involved with cæcal worms, we have recently advised inquirers to give one teaspoonful of tobacco dust in each pint of early morning mash on two or three occasions, and a teaspoonful of hydrochloric acid (B.P.) in each quart of drinking water.

Herms and Beach recommend that worm-sick land should be disinfected by swilling with 1 in 1,000 sublimate solution. Since the eggs lie not more than 5 cm. deep in the ground, a sufficient penetration of the earth with the disinfectant must be made. When the ground has completely dried out the birds may again be let on the disinfected ground. Worm-free animals should only be allowed on this ground. Using other methods, salt may be strewn on the ground and washed into it, or a 20 per cent. salt solution may be sprayed on the ground. These procedures destroy nematode eggs and larvæ of fowls. In the case of turkeys an effectual treatment is often hampered and turned to naught by the careless methods of would-be rearers. The turkey is a valuable bird, and pays for proper and skilled attention. In Ireland and Norfolk a race of people exists that have bred turkeys with success for years. There the extensive range so admirable for turkey breeding exists.

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SALMONELLA INFECTIONS IN YOUNG DUCKLINGS AND DUCKS' EGGS.

By G. H. WARRACK, B.Sc., and T. DALLING, M.R.C.V.S.
Wellcome Physiological Research Laboratories, Beckenham, Kent.

WE have been interested during the past few years in outbreaks of infectious disease among young ducklings associated with member of the Salmonella group or organisms, and have shown that such outbreaks may be caused by *S. aertrycke* and *S. enteriditis* (*gaertner*). We have investigated, further, the source of such infections, and our observations and experiments lead us to conclude that two sources are possible: (1) the Salmonella may be transmitted through the egg from infected adult laying ducks in a manner similar to the transmission of *S. pullorum* in B.W.D. among chicks; and (2) healthy ducklings may acquire infection from outside sources. We have already recorded the results of some of our findings (1931, 1932 and 1933). This paper deals with some further experiments.

Infection from Laying Ducks.

Scott (1930) discussed food poisoning in human beings attributable to the consumption of ducks' eggs infected with *S. aertrycke*, and showed experimentally that from eggs soaked in an emulsion of the micro-organism it was possible to recover cultures from the majority of eggs so treated. He further suggested the probability that in some cases the source of infection in eggs was the ovary, which may be infected as part of a general Salmonella infection. Hole (1932) reported the results of his investigations in Salmonella infections in ducklings, and showed that in one outbreak an organism apparently identical with *S. enteriditis* was isolated, while in two other outbreaks organisms closely allied to *S. aertrycke* were found. He inquired into the possibilities of egg transmission of these organisms, and though he was unable to prove the point definitely, his results indicate a distinct possiblity of egg transmission.

The problem of whether healthy-looking ducks could lay infected eggs was first solved by our investigations recorded in 1932, when we wrote: "Ducks do harbour micro-organisms of the Salmonella group, the ovary is often affected, such ducks may lay infected eggs, such eggs often fail to hatch, young ducklings may suffer from such infections in an epidemic form and the agglutination test will detect infected ducks."

Working in collaboration with Dr. W. M. Scott, we showed that ducks laying eggs infected with *S. aertrycke* or *S. enteriditis* (*gaertner*) produced in their serum agglutinins, as shown by the ability of the serum to cause agglutination of the respective antigens, and that the titres of the serum of individual ducks varied from 1 in 50 to 1 in 3,000. We were able to obtain some reacting ducks, and kept them at the laboratory for twelve months, during which time samples of their serum have been tested periodically, and all eggs laid have been examined bacteriologically.

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S. aertrycke.—Among the *S. aertrycke* reactors were twenty ducks from the same flock. They were "in lay" when we received them in June, 1932, and they continued to lay from June to August, during which time 303 eggs were produced. From eight eggs only were cultures of *S. aertrycke* obtained. In October, 1932, agglutination tests showed that all now reacted at a titre much lower than that originally recorded in June, *e.g.*, one duck's reacting titre fell from 1 in 3,000 to 1 in 300. Since October, 1932, 436 eggs have been laid, and with the possible exception of one, none have yielded cultures of *S. aertrycke.* Tests carried out at intervals over the period have shown that the titres have steadily fallen till at the last test the average titre was very low. In all, during the period of observation, six ducks have died. In one only, which died while its titre was still 1 in 40, was *S. aertrycke* recovered from the ovary.

During 1932, 3,000 samples of blood obtained from ducks on thirty different farms in different parts of the country were examined. In one only was there a positive agglutination reaction (1 in 320). This duck was sent to us. Its agglutinating titre has now fallen to 1 in 20, and on no occasion have we found *S. aertrycke* in any of its eggs.

The interesting points in connection with these observations are that it was only when the titres were high that infected eggs were laid, and the gradual dropping in titre of the reacting ducks till many became negative. With *S. aertrycke* infection it seems probable that ducks become infected temporarily, and that the ovary is one of the tissues affected. When gross infection is present, as shown by the high agglutination titre of the serum, transmission of infection may occur through the egg, but as the infection passes off, as shown by the drop in the agglutination titre, the ovary becomes cleared from infection, and the danger of laying infected eggs is over.

S. enteriditis (*gaertner*).—Concerning *S. gaertner*, we have also shown that ducks may lay infected eggs, and that the agglutination (blood) test will detect the carriers of the infection. As an example of this, the history of an outbreak which caused heavy losses, and with which we dealt, may be of interest. *S. gærtner* was isolated from the tissues of several young ducklings. On blood-testing the breeding stock, several reactors were found, and five of them were presented to us. They laid 166 eggs during the short period of observation, and from seven *S. gaertner* was isolated. All five ducks were killed, and all showed infection of the ovary with *S. gaertner.*

The same 3,000 samples of duck blood referred to above were tested by the agglutination method for the presence of *S. gaertner* infection in the ducks yielding the blood. On eleven farms reactors were found. A number were presented to us, and we have kept them under observation at the laboratory for over a year, testing their serum periodically, and examining all eggs laid. The ducks were kept in groups:

(1) From farm "A," five reactors were obtained. Two died while their titres were high, and from the ovaries of both *S. gaertner* was recovered. The

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remaining three gradually dropped in their reacting titres. While the titres of the birds were high, from seven eggs *S. gaertner* was recovered from two, but since the titres fell no infection has been found in the fourteen eggs produced.

(2) We received seven reactors from farm "B." Their titres remained steady for some months, during which time ninety-one eggs were produced; from ten *S. gaertner* was isolated. (One duck laid six eggs, and from four cultures of *S. gaertner* were obtained.) Two birds died, and the ovaries of both were infected with *S. gaertner*. The remaining birds dropped in their reacting titre, and laid no more eggs; we still have the birds.

(3) From several farms twenty-eight reacting ducks were obtained; their titres gradually fell, and at the same time there was a decrease in egg-laying; birds which died when the titre was high were proved to be infected with *S. gaertner*; no infection of eggs was shown. The interesting point in connection with these *S. gaertner* infections is the gradual fall of the degree of reaction, as judged by the serum titre at which agglutination occurred, and the apparent co-relation between the degree of reaction and the laying of infected eggs.

Infection from Outside Sources.

During the past year we have had evidence that infection can be introduced into broods of young ducklings from outside sources, and that heavy losses may take place. We were consulted in connection with losses among young ducklings on several occasions. A short history of some of the outbreaks may prove of interest.

S. aertrycke.

Outbreak A.—In June, 1932, we showed that many deaths among young ducklings were caused by *S. aertrycke*. In October, 1932, the whole of the breeding stock was blood tested, with entirely negative results. In December, 1932, a further *S. aertrycke* outbreak occurred, the source of which was probably eggs purchased from an outside source. At this time the farmer moved to another farm, and took with him the incubator in which the infected ducklings had been hatched. The incubator was not disinfected. Early in 1933 eggs from the above blood-tested stock, as well as some eggs from another blood-tested stock, were hatched in this incubator. Losses of 50 per cent. from *S. aertrycke* infection occurred among both lots of ducklings. We visited the farm and came to the conclusion that the infection was present in the incubator, which had become infected from the eggs from the outside source hatched in December, 1932, and that the ducklings became infected soon after they were hatched, though we could not recover *S. aertrycke* from the shells of many eggs examined. Our conclusions were based on the following bits of information:

(1) Ducklings brought from an outside source and kept in adjoining pens remained healthy. (2) The eggs from the outside "clean" farm hatched with

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the owner's eggs produced ducklings which died from *S. aertrycke* infection. (These ducklings were removed in clean boxes on the day of hatching, and kept on the "clean" farm. At the same time eggs from the same ducks had been hatched in the owner's own incubator; the ducklings remained healthy, though kept in compartments of the brooder, separated from the infected ducklings by a wooden partition only.) (3) We were unable to find *S. aertrycke* in any of the 108 eggs received from the infected farm. (4) We could not recover *S. aertrycke* from the shells of many eggs taken from the incubator, and from which infected ducklings had hatched. (5) We carried out full bacteriological examinations on two sick ducks from the farm, and found no trace of the infection. (6) None of the breeding stock reacted to the blood test in October, 1932. (7) The whole incubator, including the feltings, was thoroughly disinfected, and later hatches of ducklings from eggs from the same stock remained healthy.

Outbreak B.—A duck breeder experienced heavy losses among his young ducklings in December, 1932, 200 to 300 dying between the fourth and fourteenth day after hatching. We diagnosed *S. aertrycke* infection, visited the farm, and found that the breeding stock consisted of 300 birds, all apparently in good health. We advised that they be blood tested. No "positive agglutination reactors" were found. The breeding stock was kept in a large field about half a mile from the farmyard where the incubator and brooder house were situated. Opposite the incubator and brooder houses was a pigsty which had contained some young pigs, and in which the manure was still present. The farmer was advised to cleanse and disinfect the incubator thoroughly, and use a second brooder house which he had available, after its disinfection. No losses occurred in hatches subsequently reared. We concluded from this experience that here again the infection could not be traced to the breeding stock, but had arisen from some outside source. (It is well known that pigs harbour *S. aertrycke*, and the possibility exists that the infection was derived from them.)

S. gaertner.—None of the outbreaks with which we have had to deal has been shown to be caused by *S. gærtner* derived from an outside source. In connection with *S. gaertner* infections among ducklings, our observations lead us to believe that such infection is the cause of much "dead in shell" among hatching ducks' eggs. Our recommendations, based on the above results, are that when a heavy mortality among ducklings occurs, and is shown to be caused by *S. aertrycke* or *S. gaertner*, blood testing should be carried out in the breeding flock to show whether the infection is derived from them. Failing positive results, other sources should be considered.

Conclusions.

(1) Losses among young ducklings may be caused by *S. aertrycke* and *S. enteriditis* (*gaertner*). (2) Infection of ducks' eggs with *S. aertrycke* and *S. enteriditis* (*gaertner*) has been found on many occasions. (3) One source of infection is the infected ovary of the duck producing the eggs. (4) Such ducks usually show a high agglutinating titre during the period when infected eggs are being produced.

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The titre falls fairly rapidly, and coincidentally no more infected eggs are laid. (5) Infection of ducklings from outside sources also occurs.

We have been greatly assisted in these investigations by members of the Utility Duck Club and the Scientific Poultry Breeders' Association, who have collaborated with us, supplying infected birds and placing their flocks at our disposal.

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THE N.V.M.A. CONGRESS.

THE Annual Congress of the N.V.M.A. was held, from September 18th to 22nd, at Llandudno, where well over 200 veterinarians, their ladies and friends, assembled, under the presidency of Lieut.-Colonel P. Simpson, D.S.O., T.D., F.R.C.V.S., to discuss the various papers presented and to meet together socially. There can be no doubt that everyone was amply rewarded for their attendance, for all the arrangements made by those whose duty it was to " run " the Congress were well up to the standard which one has been taught to expect at the annual Congress. The " papers " went well, and created so much discussion that in most cases the session allotted to the particular subject seemed much too short. The committee who selected the subjects for discussion, and the writers and openers of the papers, showed great foresight in their choice, which met with and deserved general approval. One of the most interesting and instructive parts of all congresses is the expressionof opinions by those who have first-hand information on the various subjects, backed up by practical experience. It was also very instructive to hear the views of such well-known and well-informed gentlemen as Sir W. Dalrymple-Champneys, of the Ministry of Health, and Dr. Crowther, of the Harper Adams Agricultural College. The commercial firms displayed their goods on tastefully arranged stalls ; the social side was well catered for ; the annual banquet was as gay as ever, and the dance following it appeared to be much appreciated, as was the dance held the following evening under the auspices of the Ladies' Guild, whose efforts on behalf of our less fortunate brethren seems never ending. The President's reception and dance will be long remembered, as will the tour of Snowdonia and the happy time spent in Bangor. (The Bangor trip owes its origin to the efforts of Dr. Montgomerie.) A most interesting practical demonstration was given in the abattoir by Mr. Burrows, and the use of the electro-lethal apparatus as a means of the humane slaughtering of animals for food must have impressed all who witnessed the demonstration, and it is hoped will influence all in the position to advocate and press for its general adoption throughout the country.

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The last few stages of the Congress saw the induction of the new President, Mr. W. Nairn, M.R.C.V.S., Blairgowrie. It was with a feeling of regret that we saw our well-tried President (Lieut.-Colonel Simpson) hand over his gown and chain, but we were consoled by the fact that they will be worn for the next year at least by so worthy a member of the profession as Mr. Nairn, whose interest in the welfare of the profession is known not only in his native country but throughout the Empire.

R.A.V.C. DINNER.

R.A.V.C.—The Annual Dinner will take place at Anderton's Hotel, Fleet Street, London, E.C.4, on Friday, November 3rd, 1933, at 7.30 p.m. Tickets: members 4*s*., non-members 5*s*. A.G.M. same place and date, 6.30 p.m. Apply hon. secretary and treasurer, c/o R.A.V.C., Records Office, The Common, Woolwich, S.E. 18.

CANINE INSURANCE.

We would draw the attention of our readers to the advertisement pages, wherein they will find an interesting announcement of the Canine Insurance Company. This Company provides insurance for every dog. There is a provision for veterinary advice up to a certain amount for treatment in the case of an accident to the animal, and a fixed amount in the event of death through an accident. The premium is, we understand, only four shillings per annum, which will bring it within the reach of practically every dog-owner. The Company invite the co-operation of all members of the profession in this venture.

ERRATA.

In our September issue, in Messrs. Montgomery and Rowlands' article on "Pulpy Kidney Disease of Young Lambs in North Wales," please note the following errata:—On page 388, second paragraph, for "in 1932" read "in 1931"; third paragraph, for "in 1931" read "in 1932." Page 391, for "Summary of 1933 Observations" read "Summary of 1932 Observations." Page 398, for "[7] Montgomerie, R. F., and Rowlands, W. T. (1932)" read "[7] Montgomerie, R. F., and Rowlands W. T. (1931)."

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